A COMPARATIVE STUDY OF ESOPHAGEAL VARICES – BANDING VS SCLEROTHERAPY

Dissertation Submitted in the

partial fulfillment of the regulations required for the award of

M.S. DEGREE

in

GENERAL SURGERY

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2016

CERTIFICATE

This is to certify that this dissertation titled "**A COMPARATIVE STUDY OF ESOPHAGEAL VARICES – BANDING VS SCLEROTHERAPY"** is the bonafide work of Dr. NAVEEN M postgraduate student in M.S. General Surgery, Coimbatore Medical College and Hospital, Coimbatore. This study was undertaken in the department of General Surgery, Coimbatore Medical College and Hospital during the period June 2013 to August 2015 in partial fulfillment of the requirement of the "The Tamil Nadu Dr.M.G.R.Medical university" for the award of M.S. Degree in General Surgery. This dissertation has not been submitted in part or fully to any other University or Board. It gives me great pleasure to forward this dissertation.

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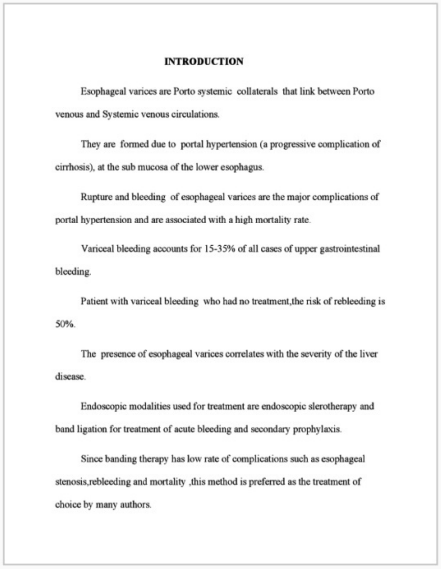


INTRODUCTION

Esophageal varices are Porto systemic collaterals that link between Porto venous and Systemic venous circulations.

They are formed due to portal hypertension (a progressive complication of cirrhosis), at the sub mucosa of the lower esophagus.

Rupture and bleeding of esophageal varices are the major complications of portal hypertension and are associated with a high mortality rate.

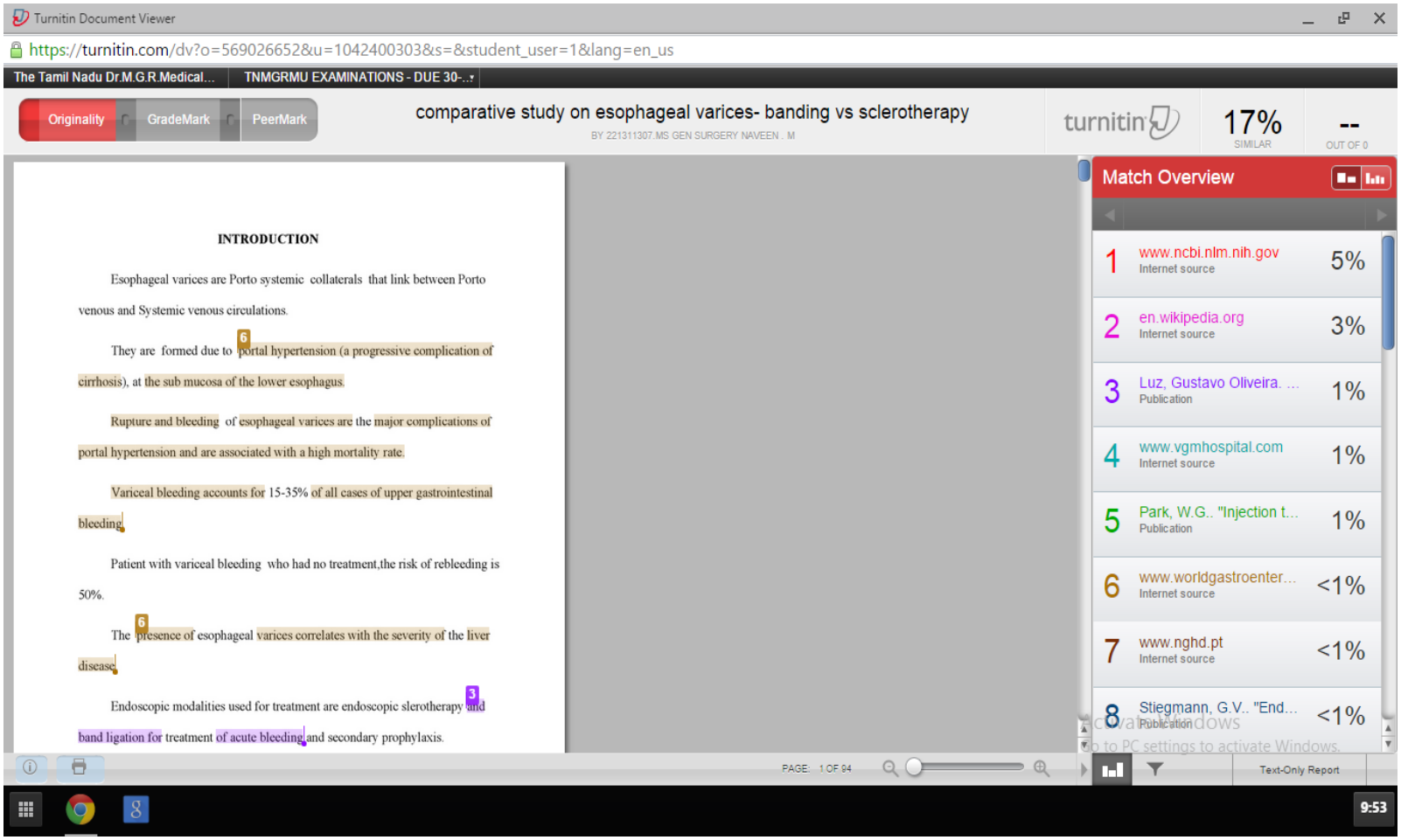
Variceal bleeding accounts for 15-35% of all cases of upper gastrointestinal bleeding.

Patient with variceal bleeding who had no treatment,the risk of rebleeding is 50%.

The presence of esophageal varices correlates with the severity of the liver disease.

Endoscopic modalities used for treatment are endoscopic slerotherapy and band ligation for treatment of acute bleeding and secondary prophylaxis.

Since banding therapy has low rate of complications such as esophageal stenosis,rebleeding and mortality ,this method is preferred as the treatment of choice by many authors.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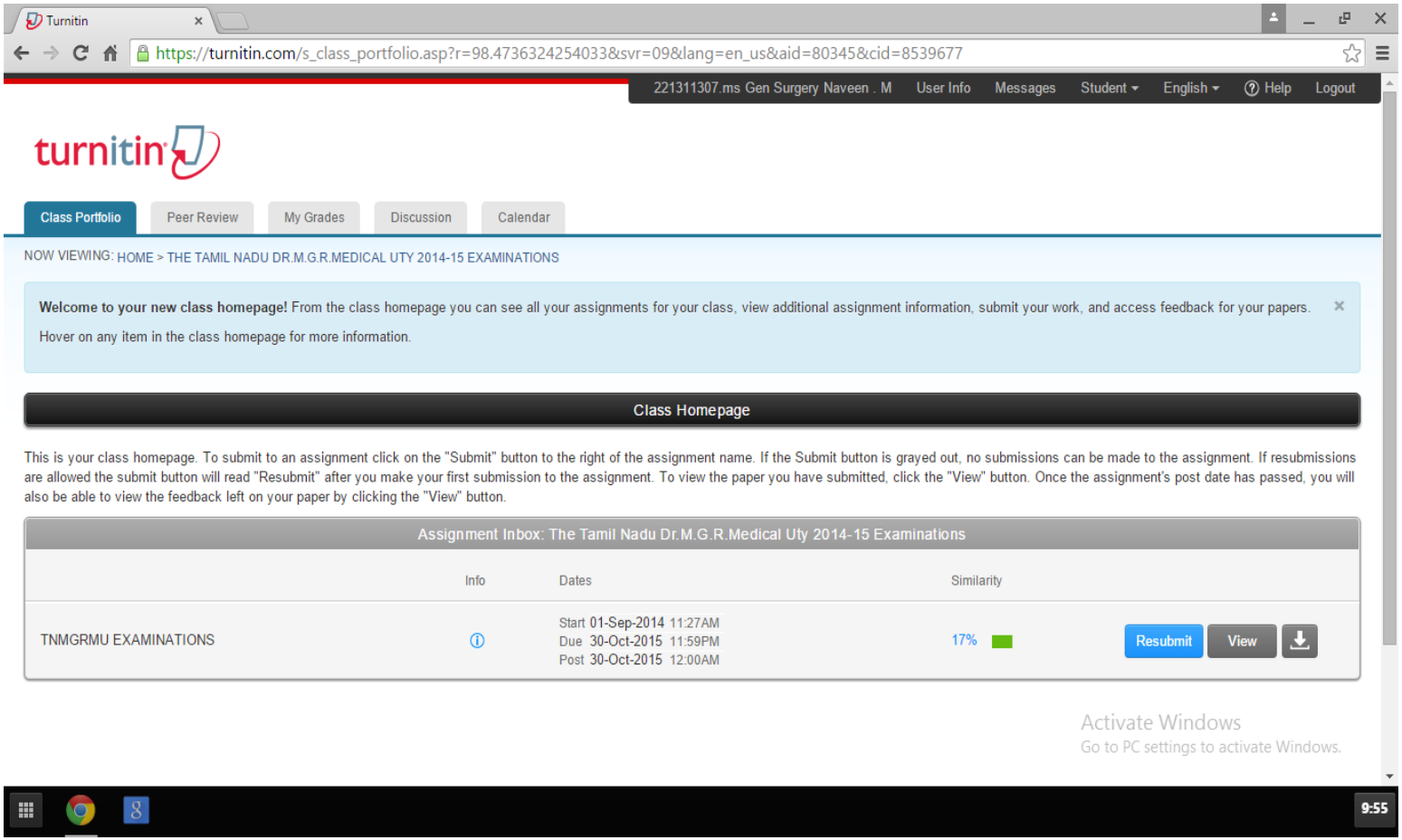
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TABLE OF CONTENTS

S.NO	TITLE	PAGE NO.
1	INTRODUCTION	1
2	AIMS AND OBJECTIVES	3
3	REVIEW OF LITERATURE	4
4	MATERIALS AND METHODS	57
5	RESULTS	63
6	DISCUSSION	78
7	SUMMARY	87
8	CONCLUSION	90
	BIBLIOGRAPHY	
	MASTER CHART	

LIST OF TABLES

S.NO	TABLES	PAGE NO.
1.	Banding and sclerotherapy in bleeding varices	65
2.	Number of bands and amount of sclerosant used	67
3.	Comparison of banding and sclerotherapy	69
4.	Comparison of retrosternal pain in banding and sclerotherapy	70
5.	Comparison of odynophagia in banding and sclerotherapy	71
6.	Comparison of fever in banding and sclerotherapy	72
7.	Comparison of tachycardia in banding and sclerotherapy	73
8.	Comparison of esophageal ulcer in banding and sclerotherapy	74
9.	Comparison of stricture in banding and sclerotherapy	75
10.	Comparison of rebleeding in banding and sclerotherapy	76
11.	Comparison of failure in banding and sclerotherapy	77

LIST OF FIGURES

S.NO	FIGURES	PAGE NO.
1.	Layers of esophagus	8
2.	Esophageal wall	9
3.	Muscularis propria	10
4.	Gastro esophageal junction	11
5.	Arterial supply of esophagus	13
6.	Venous supply of esophagus	14
7.	Lymphatic drainage of esophagus	16
8.	Nerve supply of esophagus	17
9.	Development of esophagus	17
10.	Anatomy of Portal vein	22
11.	Edoscopic sclerotherapy	33
12.	Endoscopic banding	48
13.	Sex distribution of study population	63
14.	Age and sex distribution of study population	63
15.	Age and variceal grade distribution of study population	64
16.	Randomization of actively bleeding gradeIII varices for banding and sclerotherapy	65

17.	Randomization of actively bleeding grade IV varices for banding and sclerotherapy	66
18.	Randomization for actively bleeding varices for banding and sclerotherapy	66
19.	Number of band used	67
20.	Amount of sclerosant used	68
21.	Comparison of retrosternal pain in banding and sclerotherapy	70
22.	Comparison of Odynophagia in banding and sclerotherapy	71
23.	Comparison of fever in banding and sclerotherapy	72
24.	Comparision of tachycardia in banding and sclerotherapy	73
25.	Comparison of esophageal ulcer in banding and sclerotherapy	74
26.	Comparison of esophageal stricture in banding and sclerotherapy	75
27.	Comparison of rebleeding in banding and sclerotherapy	76
28.	Comparison of failure in banding and sclerotherapy	77

INTRODUCTION

Esophageal varices are Porto systemic collaterals that link between Porto venous and Systemic venous circulations.They are formed due to portal hypertension (a progressivecomplication of cirrhosis), at the sub mucosa of the lower esophagus.Rupture and bleeding of esophageal varices are the major complications of portal hypertension and are associated with a high mortality rate.Variceal bleeding accounts for 15-35% of all cases of upper gastrointestinal bleeding.Patient with variceal bleeding who had no treatment,the risk ofrebleeding is 50%.The presence of esophageal varices correlates with the severity of the liver disease.Endoscopic modalities used for treatment are endoscopic slerotherapy and band ligation for treatment of acute bleeding and secondary prophylaxis.

AIM AND OBJECTIVES

To compare the efficacy and safety of endoscopic variceal band ligation and sclerotherapy in the management of variceal bleeding due to portal hypertension.

MATERIALS AND METHODS

The study was conducted in the Department of General Surgery in collaboration with the Department of Medical Gastroenterology and Department of Vascular Surgery, Coimbatore medical College Hospital

from June 2013- August 2015. This study was approved by the ethical committee of Coimbatore Medical College Hospital.

STUDY POPULATION:

50 patients with portal hypertension who were admitted during the study period of June 2013 - august 2015 in medicine and surgery wards, with the complaints of hematemesis and/or malena, who had grade 3 and 4 varices without gastric varices and other causes of upper GI bleeding in upper GI endoscopy were included in this study.

RANDOMISATION:

Every alternative patients presenting with above history is divided into 2 groups. One group is treated with esophageal banding and other group is treated with 3 % Sodium tetradecyl sulphate after getting informed and written consent from the patient.

INCLUSION CRITERIA:

- Age 21-70 years
- Both sexes
- Grade III and IV esophageal varices
- Patients complaining with hematemesis and/or malena
- Due to Portal Hypertension

EXCLUSION CRITERIA:

 Age <21 and >70

 Grade I and II varices

 Non portal hypertension causes of upper GI bleeding

 Prior history of endoscopic treatment and shunt operation for varices

 Presence of Hepatic Encephalopathy, Hepatorenal syndrome and life expectancy less than 48 hours

 Patients with positive serology for Hepatitis B (HbsAg) and C virus (anti HCV)

CONCLUSION

 Both banding and 3% sodium tetradecyl sulphate are equally effective in controlling acute variceal hemorrhage among which sclerotherapy had a small advantage and also in preventing rebleeding.

 Both banding and sclerotherapy are effective in eradicating varices but banding is more efficacious

 Both banding and sclerotherapy have their side effects but sclerotherapy has more frequent and dreaded complications.

 Hence banding is superior to sclerotherapy both in efficacy and safety

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Endoscopic modalities used for treatment are endoscopic slerotherapy and band ligation for treatment of acute bleeding and secondary prophylaxis.

Since banding therapy has low rate of complications such as esophageal stenosis, rebleeding and mortality ,this method is preferred as the treatment of choice by many authors.

Thereby in this study we compare the results of endoscopic banding and sclerotherapy with 3% sodium tetradecyl sulphate in the management of esophageal varices.

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REVIEW OF LITERATURE

ESOPHAGEAL VARICES:

Esophageal varices is defined as dilated sub mucosal veins in the lower third of the esophagus. They are most often due to portal hypertension, caused by cirrhosis. Normal portal pressure is approximately 9 mmHg. If the portal pressure rises above 12 mmHg, this gradient rises to 7-10 mmHg at the inferior vena cava(normal 3-7mmhg). A gradient greater than 5 mmHg is considered portal hypertension. At gradients even greater than 10 mmHg, blood flow through the hepatic portal system is redirected from the liver into areas with lower venous pressures. That means the collateral circulation develops in the lower esophageous. The small blood vessels in these areas become distended, becoming more thin- walled, and starts to bleed due to high pressure leading to esophageal varices.

GRADING (CHILD's CRITERIA):

I. Visible veins but not elevated

II. Large and raised veins but not touching each other

III. Raised and tortuous almost touching each other

IV. Very large veins touching each other

ANATOMY OF ESOPHAGUS:

It's a muscular tube of approximately 25 cm long, occupying the posterior mediastinum. It extends from upper esophageal sphincter (cricopharyngeous muscle)at the level of C6 to the junction of the cardiac of the stomach at the level of T11 thoracic vertebra and descends along the vertebral column. It also presents with flexures corresponding to the curvature at the cervical and thoracic vertebral column which is the narrowest part of the esophagus.

RELATIONS:

CERVICAL:

Anterior:

- Trachea
- Thyroid gland

Posterior:

- Longus colli muscle
- Vertebral column

On Left:

- Thoracic duct

On either side:

- Part of lobes of Thyroid gland
- Common carotid artery

THORACIC:

Anterior:

- Trachea
- Aortic arch
- Pericardium0
- Left bronchus

Posterior:

- Longus colli muscle
- Vertebral column
- Hemiazygous vein
- Right Posterior intercoastal arteries
- Thoracic duct

Right:

- Azygous vein
- Vagus nerve
- Right Pleura

Left:

- Thoracic duct
- Left subclavian artery
- Left Recurrent laryngeal nerve
- Descending thoracic duct
- Left Pleura

ABDOMINAL PORTION:

It is about 1.5 cm and situated at the posterior surface of left lobe of liver in the esophageal groove.

HISTOLOGY:

It has four layers

Mucosa

Submucosa

Muscle layer

Outer fibrous layer

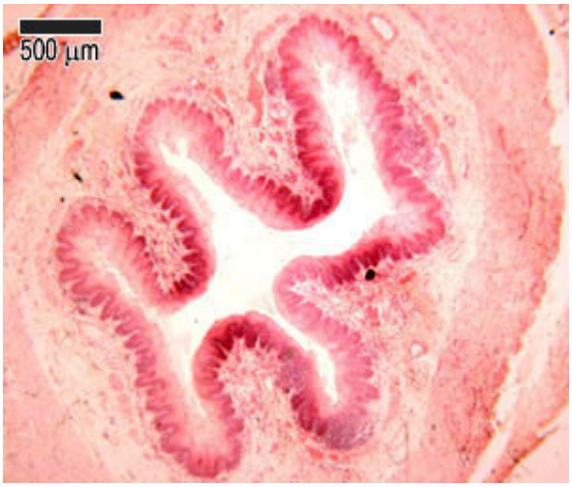

Fig.1: Layers of Esophagus

MUCOSA:

- Epithelium
- Basement membrane
- Lamina propria
- Muscularis mucosa

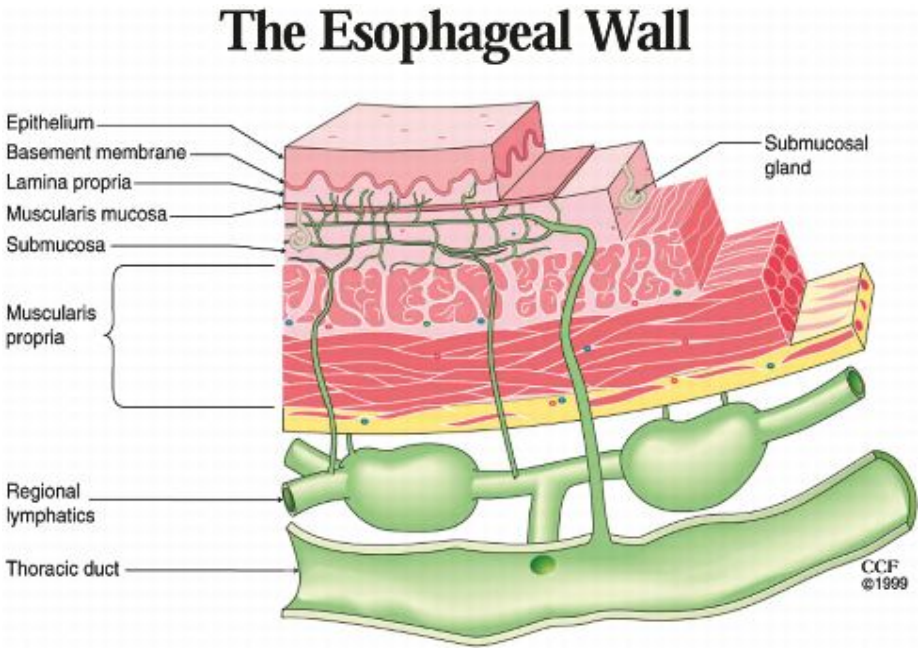

Fig2: Esophageal Wall

Lamina propria-contains lymphatic capillaries, blood vessels and loose connective tissue.

Muscularis mucosa- thin,double layered mostly abundant at the lower part of the esophagus

Z LINE:

Is the transition of esophageal mucosa to columnar epithelium at the distal 2cm of the esophagus.

SUBMUCOSA:

It is highly vascular and has loose connective tissue. It is a pavement for malignancies since it is highly vascularised and also has lymphatic structures with Meissner's neural plexus. This is the layer were dilatation of veins in esophageal varices occur.

MUSCULARIS EXTERNA:

Top third-Skeletal muscles

Middle - smooth and Skeletal muscle

Lower third-Smooth muscle

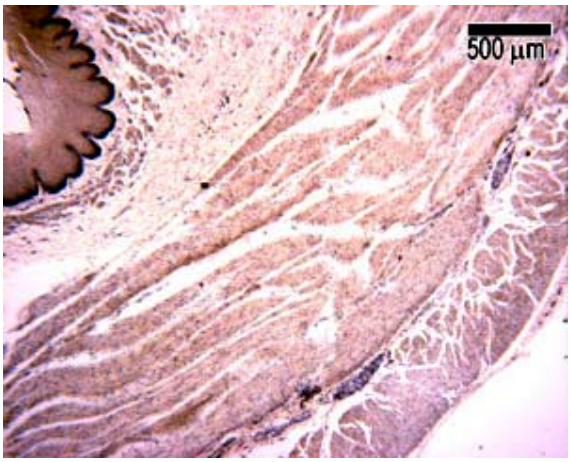

Fig3: Muscularis Propria

It consists of two layers:

- Outer Longitudinal
- Inner Circular

Auerbach's or Myentric plexus is present between the muscle layers.

GASTRO ESOPHAGEAL JUNCTION:

It is a complex valve consisting of smooth muscles(LES) and diaphragmatic element. It maintains competence during static condition and also during dynamic stress.

It has four anatomic points:

- 2 Endoscopic
- 2External

Endoscopic – Z Line

Transition from smooth muscle to rugae of stomach

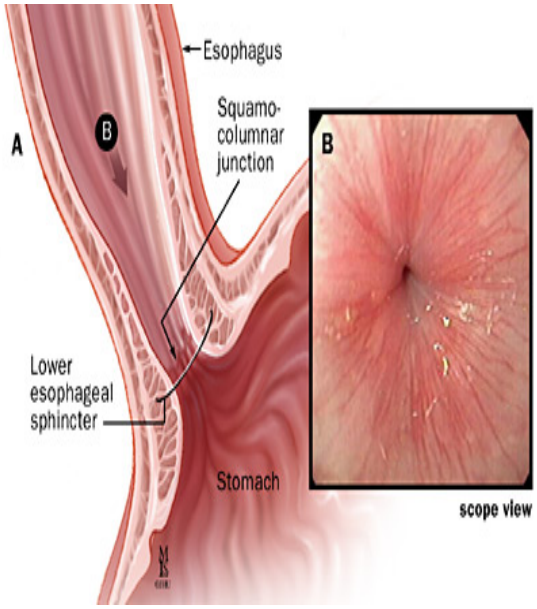

Fig4: Gastroesophageal Junction

Externally:

- Collar of Helvetius(Willis Loop)- where circular fibres of esophagus joins with oblique fibres of stomach
- Gastro Esophageal fat pad

BLOOD SUPPY:

Inferior Thyroid Arteries: Cervical Esophagus

The cervical esophagus is supplied by the paired inferior thyroid arteries. They arise from the thyrocervical trunk of the subclavian artery. The inferior thyroid arteries give off branches 2 cm to 3 cm long called tracheoesophageal arteries. These travel caudal and medial on each side toward the tracheoesophageal groove

Tracheobronchial and Bronchoesophageal Arteries: Intrathoracic Esophagus

The intrathoracic esophagus receives blood from two sources, the unpaired tracheobronchial arteries, which arise as a group from the concavity of the aortic arch and can number between one and four; and the bronchoesophageal artery.

Aortic Proper Esophageal Artery: Intrathoracic Esophagus

Left Gastric and Splenic Arteries: Abdominal Esophagus

The abdominal esophagus and gastric cardia are supplied by the unpaired left gastric and splenic arteries.These derive from the celiac axis With as many as 11 arterial branches, the left gastric artery mainly supplies the anterior and right lateral aspects of the esophageal wall

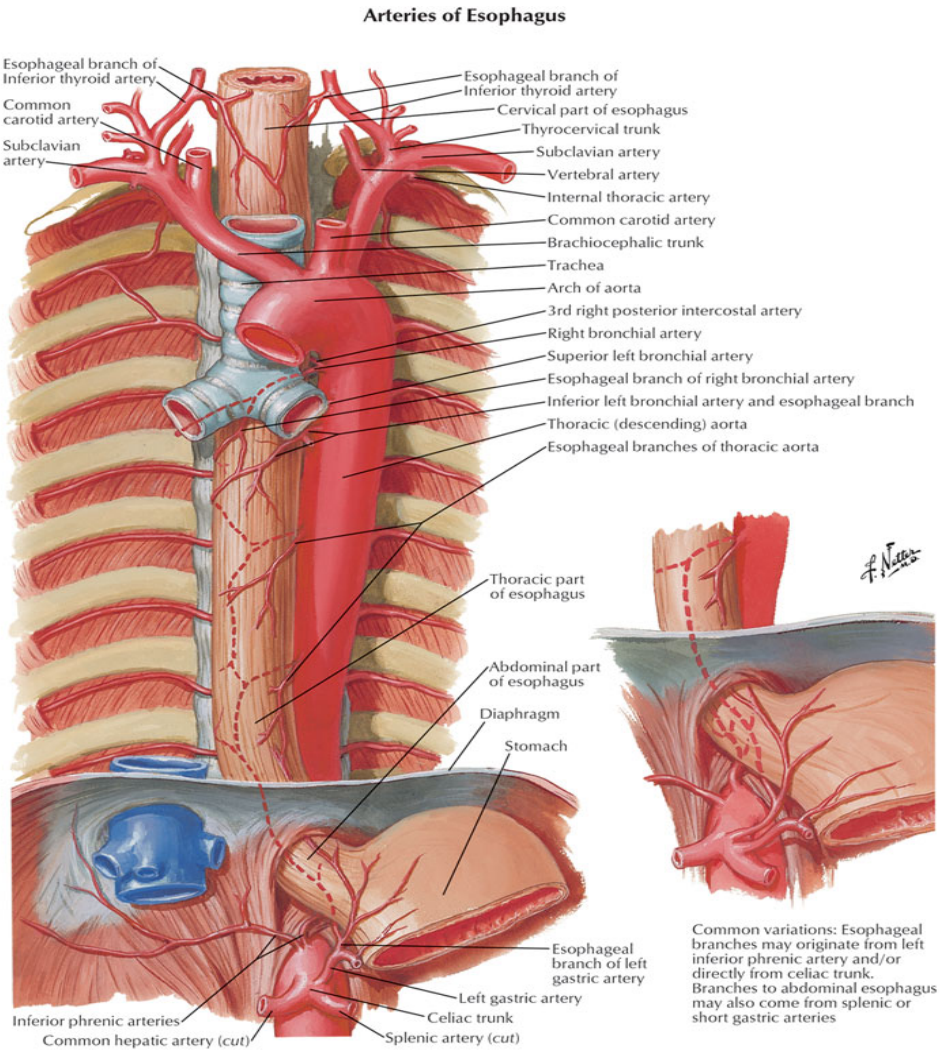

Fig5: Arterial Supply of Esophagus

VENOUS DRAINAGE:

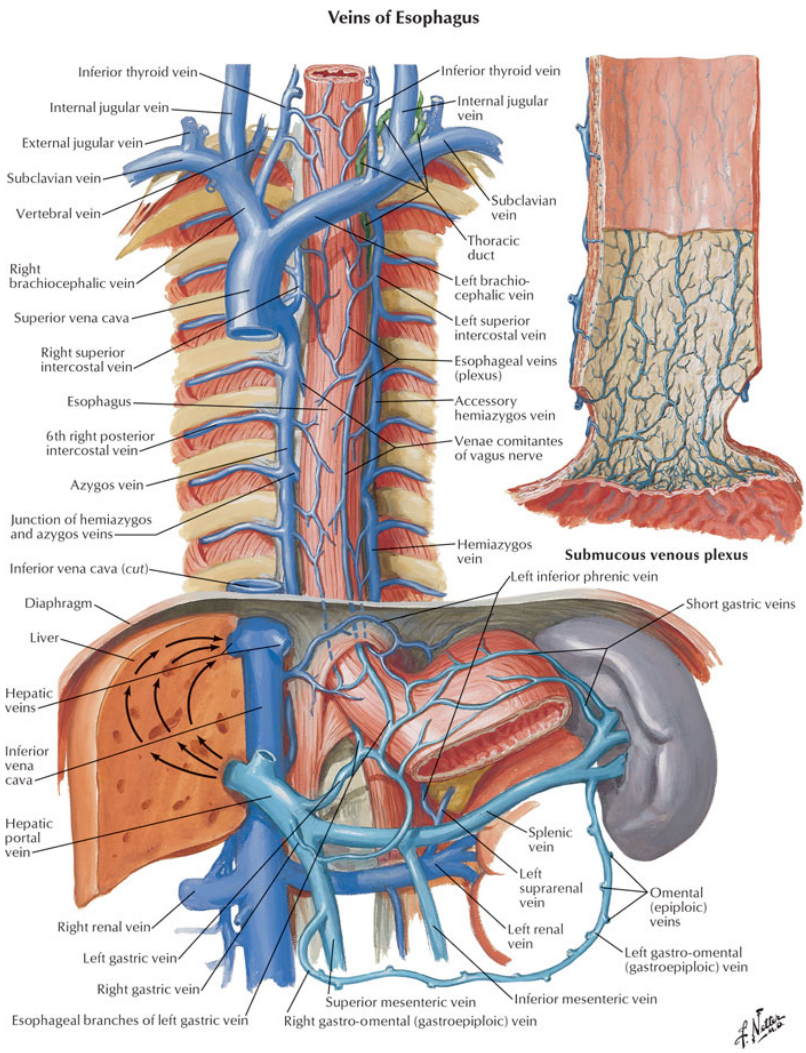

Fig**6: Venous Supply of Esophagus**

Two small veins usually accompany the circumferential arteries in the lamina submucosa. Perforating veins originating from the small communicating veins of the submucous plexus pierce the muscular wall of the esophagus together with the perforating arteries. They receive tributaries from the muscle coats and form the extramural, extrinsic veins

at the surface of the esophagus. No valves were found in the esophageal venous circulatory system.

The extrinsic veins drain into the locally corresponding large vessels. The superior vessels drain to the jugular veins or the azygos and hemiazygos veins. The inferior veins terminate in the left gastric and splenic veins.

LYMPHATICS:

The lymphatic trunks at the surface of the esophagus may drain into the regional lymph nodes. Lymph from the esophagus most likely drains into the following lymph nodes

Paratracheal
Tracheobronchial bifurcation
Juxtaesophageal
Intraaorticoesophageal

The lymph of the abdominal esophagus empties into the following lymph nodes:

Superior gastric
Pericardiac
Inferior diaphragmatic

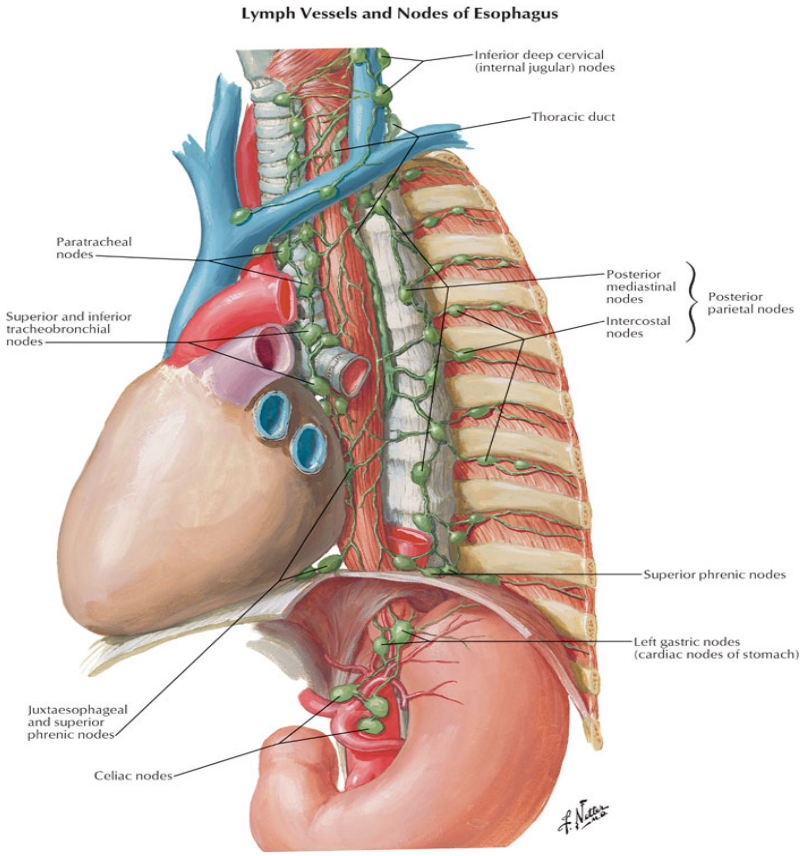

Fig7: Lympahtic Drainage of Esophagus

NERVE SUPPLY:

Innervated by both visceral components of the autonomic nervous system, the sympathetic and the parasympathetic systems, which exert mutually antagonistic influences on the viscera. The sympathetic efferent pathways, common in the gut, are concerned with vasoconstriction, contraction of sphincters, and relaxation of the muscular wall. The parasympathetic efferent fibers increase the glandular and peristaltic activity of the gut

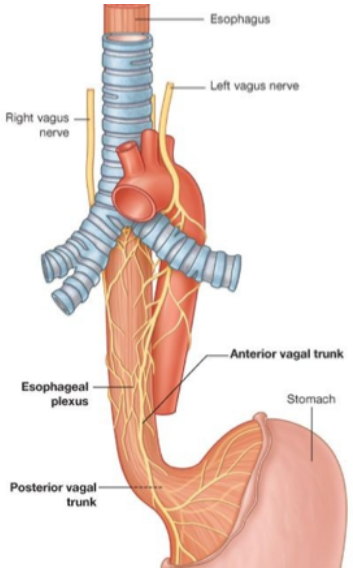

Fig8: Nerve Supply of Esophagus

EMBRYOLOGY:

In the human, the primitive foregut forms during the fourth week of gestation by a longitudinal folding and incorporation of the dorsal part of the yolk sac into the embryo.

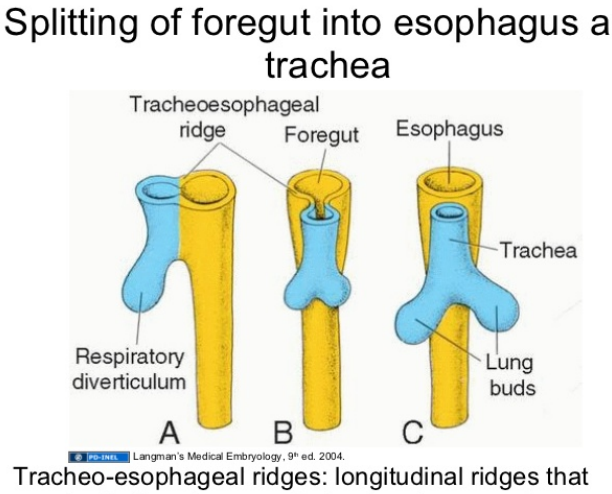

Fig9: Development of Esophagus

- ❖ Gut formation
- ❖ Gut molecular regulation
- ❖ Endodermal differentiation

In the human, the primitive foregut forms during the fourth week of gestation by a longitudinal folding and incorporation of the dorsal part of the yolk sac into the embryo. The trachea develops from the foregut about 22-23 days after fertilization as a median ventral diverticulum. Immediately after this diverticulum forms, the stomach develops further distally by an asymmetrical extension

Foregut

Several phenomena take place at approximately the 34th day. The genesis of the submucosal and muscular layers of both trachea and esophagus begins. The distal esophagus elongates first, followed by the proximal. Characteristically, the elongated esophageal segment carries the gastric-dilated primordium below the forming diaphragm. Most likely, however, elongation results from pharyngeal ascent rather than gastric descent.

Separate growth processes of the trachea and esophagus occur before the fifth week of intrauterine life. The esophagus attains its final

dimensions in the seventh week. At birth its length is 8-10 cm, which doubles in the first few years of life.

Early in the sixth week, the mesenchymal circular muscle coat develops. Three to nine weeks later, longitudinal musculature appears. During the 4th month, the muscularis mucosa appears. Blood vessels enter the esophageal wall during the seventh month, and lymph capillaries enter the wall between the third and fourth months of life after birth.[7]

At the seventh to eighth week the esophageal lumen is almost filled with cells from the proliferated esophageal epithelium. Because the filling is never complete and small vacuoles are present, the so-called solid stage does not exist as such. Around the 10th week the lumen is restored since the vacuoles coalesce.

Changes are also taking place in the esophageal ciliated epithelium, which becomes stratified squamous in the proximal and middle esophagus. Columnar epithelium remains unchanged in the distal esophagus.

The esophageal wall receives both sympathetic (thoracic trunk and celiac plexus) and parasympathetic (vagus nerve) innervation

PORTAL HYPERTENSION:

Portal hypertension is increased blood pressure in the portal vein system, which is composed of the portalvein, and its branches and tributaries. Portal hypertension is defined as elevation of hepatic venous pressure gradient to >5mmHg

Elevated pressure is because of,

- Increased pressure gradient for blood flow in liver
- Blood flowing in alternating channels

Patients develop symptoms once pressure raises above 10 mm hg

CAUSES:

PREHEPATIC:

- Portal vein thrombosis
- Splenic vein thrombosis
- Malignant occlusion
- Massive splenomegaly

HEPATIC:

PRE- SINUSOIDAL

- Schistosomiasis
- Congenital hepatic fibrosis

SINUSOIDAL

- Cirrhosis
- Alcoholic hepatitis

POST SINUSOIDAL

- Veno-occlusive disease

POST HEPATIC:

- Budd chiari syndrome
- Inferior vena caval webs
- Veno-occlusive disease
- Cardiac causes
    - Restrictive cardiomyopathy
    - Congestive pericarditis
    - Severe congestive heart failure

Portal vein drains the deoxygenated blood from instestine, stomach, pancreas, spleen and gall bladder. It is formed by splenic vein and superior mesenteric vein. Superior mesenteric vein drains the entire small bowel, ascending colon and a part of the descending colon and the head of pancreas.

Inferior mesenteric vein joins the splenic vein and hence drains the tranverse colon and part of descending colon and upper two third of the rectum. Hence the Portal vein receives blood from the entire gastrointestinal tract.

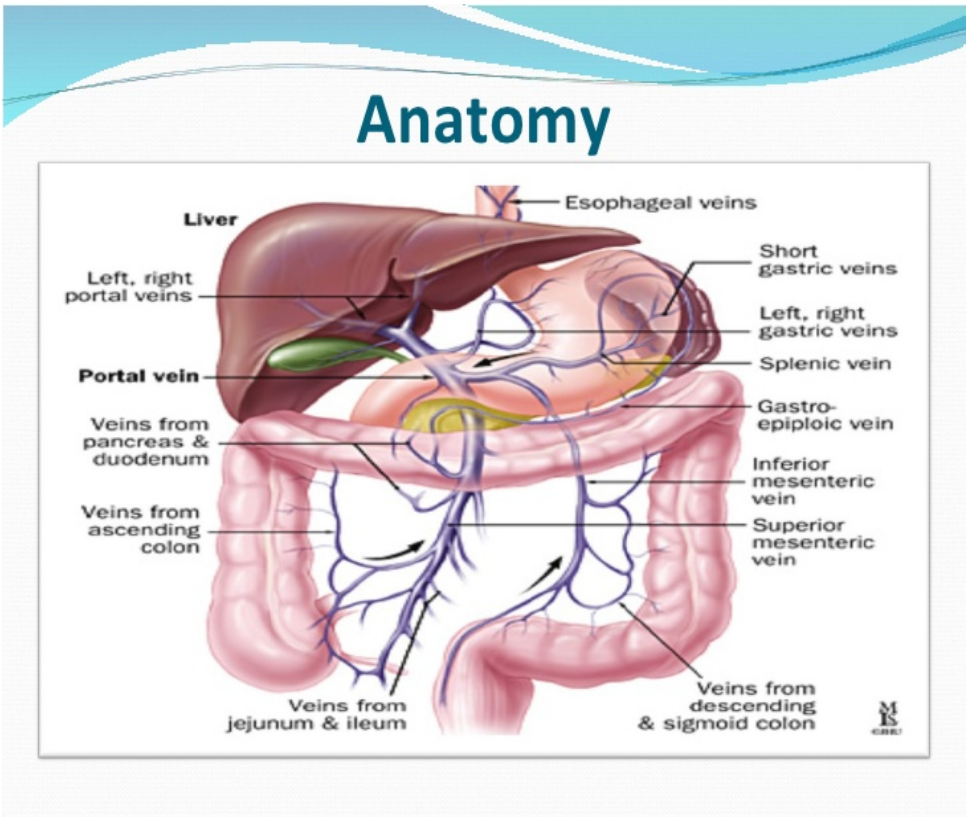

Fig 10: Anatomy of Portal Vein

Patients with pressure gradient >12 mm Hg are at high risk of variceal bleeding.

The major complication of portal hypertension is:

- Massive upper GI bleeding due to ruptured esophageal varices and portal hypertension gastropathy.
- Ascites
- Hepatorenal syndrome
- Hepatic encephalopathy

CLINICAL COURSE OF VARICEAL BLEEDING

Portal hypertension causes porto systemic collaterals development among which esophageal and gastric varices are dangerous because their rupture causes variceal hemorrhage which is the most lethal complication of cirrhosis.

When cirrhosis is diagnosed, about 25-45% of compensated patients have varices and 65% of decompensated have varices. 80% of cirrhotic patients develop esophageal varices in their lifetime and among them 35% bleed. Once cirrhosis is found, the incidence of new varices is 7% per year. Varices increases in size before they rupture and bleed.

Rate of progression of varices range from 10-35% per year. This variability is due to inter observer variability, decompensated cirrhosis (CHILD b/c), alcoholic etiology, selection of patients and presence of red spots at the esophageal varices.

The most important factors are variceal size, severity of liver dysfunction and red wale markings. The North Italian Endoscopy Club(NIEC) index allows the classification of patients into different groups with a predicted 1 year bleeding risk. According to the NIEC index, patients with small varices and advanced liver disease are at high risk of early bleeding. The probability of bleeding within 1 year in Child Pug class A patients with large varices and red sign is 24%, but in Child-Pug C patients with small varices and no red signs, Incidence of bleeding within 1 year is 25%.

Variceal size is the most useful predictor for variceal bleeding. Variceal size and red signs denote the variceal wall tension which is the factor determining variceal rupture. Risk of bleeding is very low(1-2%) in patients without varices and increases to 6% per year among patients with small varices. It increases to 18% per year in patients with medium or large varices. Red sign is another predictor of variceal bleeding. Studies have shown that variceal bleeding occur if the HPVG reaches a threshold of 12mmHg. If the HPVG is reduced (below 12mmHG or >20% of the

baseline levels), there is a marked reduction in the risk of bleeding, spontaneous bacterial peritonitis, development of ascites and death.

Variceal bleeding is the second most common of mortality among the cirrhotic patients. In cirrhotic patients, variceal bleeding causes 60% of upper digestive bleeding. Mortality from variceal bleeding has greatly decreased from 42% to 6-12% over the last two decades according to Graham and Smith study in 1981. This is due to implementation of effective endoscopic and pharmacological therapies, Trans jugular intraheapatic portosystemis shunt (TIPS) and improved general medical care. Death occurring within 6 week from the hospital admission for variceal bleeding is considered as a bleeding-related death.

Immediate mortality from uncontrolled bleeding is about 4% to 8 %. Prehospital mortality from variceal bleeding is 3%. Other causes for mortality are due to kidney failure, heapatic encephalopathy ,infection, poor liver function, severe portal hypertension with HPVG>20 mmHg and active bleeding at endoscopy.

MANAGEMENT OF VARICES:

- **Conservative**
- **Pharmacological**
- **Decompressive shunts**
- **Devascularisation procedure**

- **Endoscopic therapy**
- **Liver transplantation**

Conservative Management:

A **Sengstaken–Blakemore tube** is a medical device inserted through the nose or mouth and used occasionally in the management of upper gastrointestinal hemorrhage due to esophageal varices. The device consists of a flexible plastic tube containing several internal channels and two inflatable balloons. Apart from the balloons, the tube has an opening at the bottom (gastric tip) of the device. More modern models also have an opening near the upper esophagus; such devices are properly termed **Minnesota tubes**. The tube is passed down into the esophagus and the gastric balloon is inflated inside the stomach. A traction of 1 kg is applied to the tube so that the gastric balloon will compress the gastroesophageal junction and reduce the blood flow to esophageal varices. If the use of traction alone cannot stop the bleeding, the esophageal balloon is also inflated to help stop the bleeding. The esophageal balloon should not remain inflated for more than six hours, to avoid necrosis.

Pharmacological Therapy:

- Non cardio selective beta blocker
- Vasopressin, terlipressin
- Somatostatin analogue

NON CARDIO SELECTIVE BETA BLOCKER:

a. **Propanolol**

b. **Nadolol**

These drugs were introduced by Lebrec and his colleagues in early 1980 to reduce portal hypertension which is the mainstay of prophylactic therapy.

Advantage:

It plays a major role in preventing the initial bleed, managing acute variceal bleed and also first line in preventing rebleeding.

Disadvantages:

It had limited use in patients with Kidney disease, heart disease, astma and other lung diseases, diabetes and drug allergies.

VASOPRESSIN AND TERLIPRESSIN:

These drugs are used at acute variceal bleeding by reducing the portal pressure. Vasopressin has significant side effects with systemic vasoconstriction. So it is largely replaced by terlipressin.

SOMATOSTATIN AND ANALOGUE (OCTREOTIDE):

These are synthetic analogues of stomatostatin which act by inhibiting the release of vasodilatory hormones and also causing splanchnic vasoconstriction which in turn lowers the portal blood flow thereby decreasing bleeding and preventing rebleeding.

DECOMPRESSIVE SHUNTS:

Decompressive is mostly used as a second line and is reserved only who rebleed after endoscopic therapy and beta blockers. Surgical shunts are of 3 categories:

- Total shunt
- Partial shunt
- Selective shunt

TOTAL SHUNT:

- Classical end to side portocaval shunt
- Side to side porto caval shunt

Shunt size need to be atleast 10mm in diameter. Both are effective in controlling varices but especially end to side shunt is more effective then side to side while only the latter is effective in controlling ascites.

Disadvantages:

These shunts are associated with increased incidence of Hepatic encephalopathy.

PARTIAL SHUNT:

Shunt size is about 8mm. Polytetrafluroethylene (PTFE) interposition the grafts between portal vein and inferior venacava found to be greater than 90% control of varices and maintain portal perfusion.

SELECTIVE SHUNTS:

Distal Splenorenal Shunt (DSRS), THE WARREN SHUNT:

In patients who have impaired hepatic reserve, the Warren shunt has been proposed as an effective operation because it

decompresses the esophageal varices without disturbing portal perfusion of the liver. It is the anastomoses between splenic veins with the left renal vein after its disjunction with the superior mesenteric vein. Control of bleeding and portal perfusion is maintained in more than 95%of individuals. Incidence of hepatic encephalopathy after the shunt is around 15%.

Transjugular intrahepatic porto systemic shunt (TIPS):

TIPS were described by Rosch in 1969 but only in 1982 it was first used in humans by Dr.Ronald Calpinto. It became successful only with the development of endovascular stents in 1985. From 1988 the procedure has widely being accepted and preferred method for treating portal hypertension refractory to medical treatment. Hence TIPS widely replaced the surgical portocaval shunt. TIPS is the puncture of internal jugular vein, passage of catheter into one of the major hepatic vein(usually right) through the right atrium followed by transparenchymal liver puncture to cannulate the portal vein.

Intraparenchymal tract is dilated and is stented with an expandable metal stent. Pressure is measured before and after keeping the stent and the goal is to attain pressure difference of less than 10mm Hg between portal vein and right atrium. The success rate is high with less morbidity.

Disadvantages of TIPS are its thrombosis and restenosis which necessitates frequent repeat procedures and monitoring. The early thrombosis is related to bile duct puncture since the bile is extremely thrombogenic, occlusion occurs within first 24 hrs.

DEVASCULARISATION PROCEDURES:

These operative procedures take care of the variceal bleeding by interrupting the inflow to the varices. The **Sugiura procedure** is a surgical technique that involves the removal and transection of the blood vessels that supply the upper portion of the stomach and the esophagus. The procedure also involves a splenectomy. The original technique described by Sugiura and Futagawa was a two-step operation consisting of an initial thoracic operation followed by the abdominal operation 3–4 weeks later. The thoracic operation consists of an extensive paraesophageal devascularization up to the inferior pulmonary vein and esophageal transection. The abdominal operation consists of a splenectomy, devascularization of the abdominal esophagus and cardia, and a selective vagotomy with pyloroplasty. The advantage is that they do not reduce portal hypertension and hence maintaining portal perfusion of the cirrhotic liver. The disadvantage is relentless recollateralization of varices across the esophageous and stomach with risk of rebleeding.

LIVER TRANSPLANTATION:

Variceal bleeding per se is not on indication for liver transplantation while the associated ascites and encephalopathy are indicators of end stage liver disease and for liver transplantation.

The timing of transplant is by the severity of the underlying liver disease.

ENDOSCOPIC THERAPY:

- SCLEROTHERAPY
- BANDING
- TISSUE ADHESIVES
- ENDOLOOPS
- BALLOON TAMPONADE
- CYANOACRYLATE GLUE INJECTION

ENDOSCOPIC SCLEROTHERPY:

HISTOLOGICAL ASPECTS:

Endoscopic sclerotherapy for esophageal varices was first reported by two Swedish surgeons Crafoord and Freckner in 1939 in 19

yr old female using Quinine as sclerosant every alternate day for one month till the varices obliterated. Then in 1940, a thoracic surgeon Moensch at Mayo clinic reported the second case of sclerotherapy

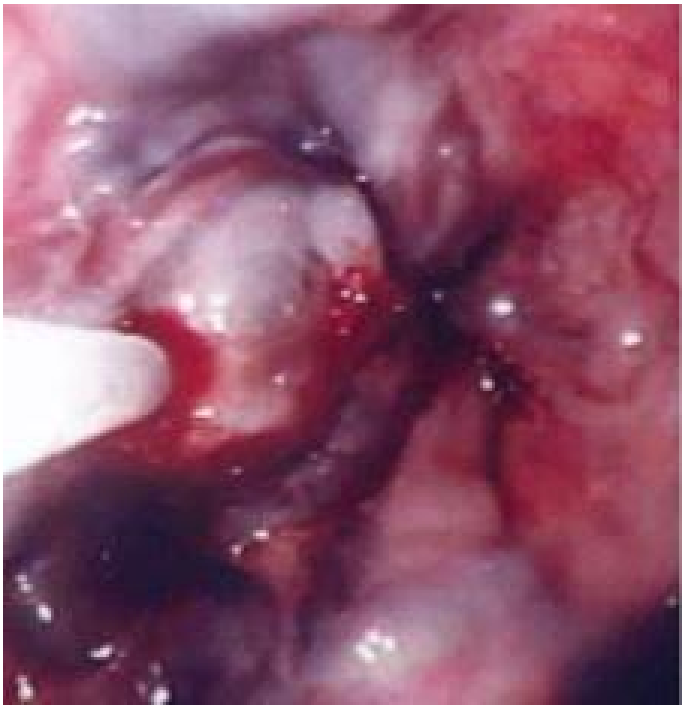

Fig 11: Endoscopic Sclerotherapy

SCLEROSING AGENTS:

Sclerosing agents were actually used in 1920 for varicose veins of lower limbs. The choice of sclerosants depends on the number of considerations including the efficacy of the agent, injection techniques, safety profile, availability and cost.

The sclerosing agents available are:

SYNTHETIC PRODUCTS

- Sodium tetradecyl sulphate
- Polidocanol

FATTY ACID DERIVATIVES

- Sodium morrhuate
- Ethonalamine oleate

OTHER AGENTS

- 3%phenol in water
- 5%phenol in oil
- Absolute alcohol

FACTORS INFLUENCING SCLEROTHERAPY:

A number of factors influence the effect of sclerotherapy on esophageal varices which includes choice of endoscope, injection sites, timing of the injection, amount and type of sclerosant used and clinical condition of the patient.

MECHANISM OF ACTION:

The mechanism of action of these agents are poorly defined but the effects involve more than simple initiation of the clotting process of intimal injur. Autopsy studies showed that the thrombosis of the submucosal vessel occur within in the first 24 hours along with tissue necrosis even in the absence of extravasations of these agents, while superficial or deep ulceration occurs after seven days. Submucosal fibrosis was seen one month of sclerotherapy.

These extravasation effects may be responsible for the long time success of sclerotherapy with the development of fibrosis preventing the formation of new variceal channels in the adjacent mucosa. Hence, procedures which are directed only at the varices often fail because of subsequent ligation of collaterals. Sclerotherapy achieves hemostasis through a tamponade effect and also by induction of local thrombosis followed by sclerosis due to sclerosant.

SODIUM MORRHUATE:

Sodium morrhuate, is sodium salt of the fatty acid in cod liver oil was first described in 1993. It is available in 5% solution. It is being less irritating to the adjacent tissues than phenol and quinine mixtures which were in use at that time. Studies shows rebleeding rate was 17%,

Ulceration were seen in 23%, fere in 28% and pleural effusion in 7% and esophagopleural fistula in 4%. Although sodium morrhuate appears to be an effective sclerosing agent the incidence of deep post sclerosis ulceration and other serious complication is clearly a restricting factor in its use.

ETHANOLAMINE OLEATE:

Ethanolamine oleate is derived from oleic acid and is similar in physical properties to sodium morrhuate. It is also available in 5% solution. Johnston and Rodgers used 5% ethanolamine oleate in their experience of 15 yers reported rebleeding rate of 7% and a mortality of 18%.

The most common complications were pyrexia which was seen in 39% and retrosternal discomfort which was seen in 30% of patients. Even though ethanolamine oleate enjoys a good reputation as a sclerosing agent, the data available at present would not appear to give this drug a clear advantage in either safety or efficacy over other agents.

ALCOHOL:

The advantage of alcohol is its easy availability and economy. The success rate in controlling the variceal bleed was 92% with rebleed rate of 32%. There is a higher complication rate with an intravariceal injection

of absolute alcohol most commonly ulcerations. Through alcohol may appear to be an effective sclerosing agent, the higher incidence of severe retrosternal pain, dysphagia, ulcers and stricture is clearly a restricting factors in its use.

PHENOL:

Supe in 1994 used 3% aqueous phenol for sclerotherapy of esophageal variceal bleeding. Preobliteration variceal bleeding appeared in 15% of the patients. Complication such as esophageal ulceration, stricture and perforation were observed in 32%, 4.5% and 1% of the patients. Complication such as esophageal ulceration, stricture and perforation were observed in 32%, 4.5% and 1% of the patients respectively. Though it is cheap and freely available, because of the high complication rate, use of phenol as a sclerosing agent was given up.

POLIDOCANOL:

Hydroxypolyethyoxydodecan (HPD) or polidocanol is commercially available as aethoxysclerol. It is a synthetic product available in uniform lots. It is marketed as 0.5%, 1,2,3% solutions. Paquet has concluded by his study that 1% polidoconal was associated with decreased rate of complication. Deep ulcerations were seen in 11 patients out of 640 patients, while superficial ulceration occurred in 30

patients, pleural effusion in 14 patients. Minor complication like retrosternal pain were observed in 15% of the patients. Sorensen *e*t al., described a higher rate of esophageal stricture (59%) with the use of 3% polidocanol especially when more treatment sessions and greater amount of scerosants were used[18].

SODIUM TETRADECYL SULPHATE:

Sodium tetradecyl sulphate was first suggested as a a sclerosing agent in 1946. Reiner noted that the agents in use at that time, such as sodium morrhuate, were soaps of naturally occurring oils and allergic reaction did occur ranging from rash to anaphylaxis. Surface activity of the fatty acid anions of the soap was believed to be physical activity responsible for thrombosis. This activity was enhanced in this synthetic anionic detergent, sodium tetradecyl sulphate.

Hence being a detergent based chemical, its action is on the lipid molecules in the cells of the vein wall watch result in the destruction of the internal lining of the vein and eventual sclerosis of the vine. It is available in the concentrations of 0.2%, 0.5%, 1.0%, 1.5%, and 3.0%. Solutions.

Sodium tetradecyl sulphate occurs as white waxy solid. Sotradecol is a sterile non pyrogenic solution of sodium tetra decyl sulphate which is

used as a sclerosing agent. These drugs are widely used for varicose veins.

ADVERSE EFFECTS:

Local reactions such as pain, ulceration are common at the site of injection. Systemic reactions reported are headache, nausea, vomiting. Allergic reactions like asthma, hay fever and anaphylactic shock were also reported. Six deaths have been reported among which anaphylactic shock accounts for four patients, one is due to asthma and the last is due to its concomitant use with anti-ovulatory agents.

Blenkinsopp showed that 3% STD was efficacious than 1% but due to damage to arterial wall was seen at both 1% and 3% concentrations but at a lesser incidence compared to other sclerosants. This is of interest because the bleeding from deep ulcerations following sclerotherapy was to be related to arterial damage rather due to portal hypertension. Post sclerotherapy ulcerations were found to be superficial ulceration. Thus sodium tetradecyl sulphate can be considered as one of the potent sclerosing agent at present.

TECHNIQUE:

There is no accepted standard technique for sclerotherapy injections. One disparity lies between paravariceal and intravariceal injection.

Intravariceal technique:

It is known as Anglo-American method. Sclerosants are directly injected into varices. All visible varicies are injected with 1-2 ml of sclerosant directly then with 1-2ml 1cm below the bleeding site. Then 1 ml of sclerosant is injected at gastro esophageal junction along all the varices. Even though the varices are present more proximally injections are placed up to 10 cm from gastro esophageal junction in 3-5 cm intervals unless a more proximal bleeding site is identified since the sclerosant can escape from varix into the azygous system and then into pulmonary circulation. Total volume of sclerosant should never exceed 20 ml per session or 5 ml per varix.

Paravaricel technique:

It is known as Europen approach. In this technique sclerosant is injected into the adjeacent submucosa of the visible varices. At first slerosant is injected at the gastro esophageal junction and it is repeated circumferentially upto 10 cm proximally in a spiral fashion. The

advantages is that it controls bleeding by causing subsequent inflammation and fibrosis around the vessel wall while preserving vessel patency allowing for portal decompression and also preventing the formatin of collateral vessels.

Sarin *et al*, used a transparent Teflon injector with a needle for injection sclerotherapy. If after puncturing the varices, blood could be seen to flow up into the Teflon injector, it was taken as intravariceal injection. He concluded that intravariceal sclerotherapy was superior to paravariceal sclerotherapy in the control of active bleeding and for total variceal obliteration but paravariceal injection is associated with low recurrence rate. Hence a combination of intravariceal and paravariceal injection is superior to the above two.

COMPLICATIONS OF SCLEROTHERAPY:

EARLY

- Dysphagia
- Low grade fever
- Retrosternal pain
- Chest radiographic changes
- Pleural effusion

DELAYED

- Perforation
- Mucosal ulceration
- Esophageal strictures
- Mediastinitis
- Pneumothorax
- Acute respiratory distress syndrome
- Fistulas
- Pericarditis
- Mesenteric venous thrombosis
- Bacteremia
- Esophageal motility disorders

Sclerotherapy is associated with a wide range of complications ranging from transient pyrexia to esophageal perforation leading to death.

Complications following slerotherapy depend upon the:

- Nature of sclerosant used
- Amount of sclerosant
- Concentration of the drug

- Injection site
- Time interval between the sessions

Minor complications like fever, retrosternal pain and dysphasia occur so frequently that these are considered as side effects and not as complications.

ESOPHAGEAL COMPLICATIONS:

Esophageal ulceration occurs frequently following emergency sclerotherapy. Sarles included the most superficial ulcers too and arrived at 65% incidence of ulceration with 1% sodium tetradecyl sulphate. Conversly, Soderlund included only the ulcers that were associated with bleeding or were deep enough to prevent further sclerotherapy and reported an incidence of 25%. Japerson reported esophageal ulceration in 77% and ulcerogenic bleeding in 15%

Following sclerotherapy with 1 % poidocanol. In the presence of deep ulcers, further injection should be deferred to prevent esophageal perforation.

Esophageal perforation is the most dreaded complication and has an incidence of 1-7 %.These patients may be managed non operatively with either enteral feeding or paraenteral hyper alimentation and a course of intravenous antibiotics. Sarles using 1.5% sodium tetradecylsulphate,

noted esophageal perforation in 0.5% patients while Jasperson reported 3% incidence of perforation with the use of 1% polidocanol.

The incidence of esophageal strictures ranges from 0.9% as reported by Johnson with the use o ethanolamine oleate to 54% with the use of 3 % polidocanol. Soren high rate esophageal stricture was attributed to the frequency and amount of sclerosant used.

Transient substernal pain may occur in 55% of patients following sclerotherapy. The pain is due to inflammatory mediatinitis or esophageal spasm. Chronic dysphagia following sclerotherapy may be due to distal esophageal strictures but 4-6% is due to impaired motility.

Edophageal manometric studies show no decrease in esophageal sphincter pressure but shows marked abnormalities in esophageal peristalsis. Esophageal carcinoma has been reported in one case after undergoing nine sessions of sclerotherapy with 3 % polidocanol.

REGIONAL COMPLICATIONS:

Pulmonary complications due to sclerotherapy range from asymptomatic changes on x ray to plural effusion, pneumonia and ARDS. Hughes reported 50% incidence of pleural effusion resolves spontaneously. Pneumonia occurs due to aspiration. The development of ARDS occurs with use of sodium morrhuate. Monroe discovered that

sodium morrhuate caused transient pulmonary hypertension associated with increased flow of protein poor lymph.

SEPTIC COMPLICATIONS:

20-30% of patients had fever lasting for 24-48 hours following sclerotherapy. When fever lasts more than 2 days, the diagnosis of sepsis should be made. Transient bacteremia was reported in 15% of patients. Pneumococcus, streptococcus and staphylococcus were the commonest organisms isolated. The risk sepsis due to sclerotherapy was not related to the amount of sclerosant used, number of sessions or the cause of the liver disease. Sandy demonstrated a threefold increase in the incidence of bacteremia if the needle was inserted up to 5-7 mm instead of 3-5 mm. The incidence of clinically evident sepsis after sclerotherapy is low, so antibiotic prophylaxis for the patients undergoing sclerotherapy those who have significant valvular heart disease.

Other septic complications such as brain abscess, spontaneous bacterial peritonitis, perinephric abscess, purulent meningitis have also been reported. Other complications such as gastric ulcers, bleeding duodenal varices, portal vein thrombosis, colonic varices and mesenteric vein thrombosis have also been reported.

ENDOSCOPIC GLUE INJECTION:

Tissue adhesives are compounds that can be used for hemostasis, wound closure, or fistula repair. The main classes of tissue adhesives are cyanoacrylate glues, fibrin glue, and thrombin. Cyanoacrylate glues are used primarily for endoscopic control of bleeding from gastric varices and less commonly for hemostasis of other bleeding lesion

The tissue glue, *N*-butyl-2-cyanoacrylate, is a watery solution that polymerizes and hardens within 20 s in a physiological milieu and instantaneously on contact with blood. Because the rapid solidification of the glue makes endoscopic application technically difficult, it is necessary to dilute it with the oily contrast agent Lipiodol Ultra Fluid (Therapex, Canada). A 50/50 mixture of Histoacryl (B. Braun Melsungen AG, Germany) and Lipiodol is used for injection.

Patients with allergies to iodine should not receive this therapy because Lipiodol is an iodized oil emulsion.

ENDOSCOPIC VARICEAL LIGATION:

Endoscopic variceal ligation was first introduced to esophageal varices in 1989 by Stiegmann and Goff. This technique is an adaptation of the similar banding ligation hemorrhoids. In contrast to sclerotherapy where chemical action is used, in variceal ligation obliteration of the

varices is caused by mechanical strangulation with rubber bands, because of its action on the suctioned, entrapped varices, the reaction is usually limited to the superficial esophageal mucosa.

Endoscopic variceal ligation is placement of the rubber ring over the variceal column which is then sucked into a plastic cylinder which is attached to the tip of the endoscope.

Previously single shot ligation was used. Multiple shot devices have replaced the previous one because of its simplicity and rapidity and also over tube is not recommended and hence preventing its serious complications related to its use. And also new transparent caps are available which improve the visibility. Several commercial multiband devices are available for EBL.Multiband devices have 4-10 bands.

TECHNIQUE:

Diagnostic endoscopy is performed and varices are identified. The distance is measured from the mouth by the markings in the endoscope. The endoscope is withdrawn and is loaded with ligation devices. Devices is firmly attached to the scope and placed in neutral mode. Endoscopy with the loading device is passed which need little experience. Slight flexion of the neck, gentle and constant advancement of the scope with a

slight torque of the shaft right and left with visualization of the pharynx would guide it.

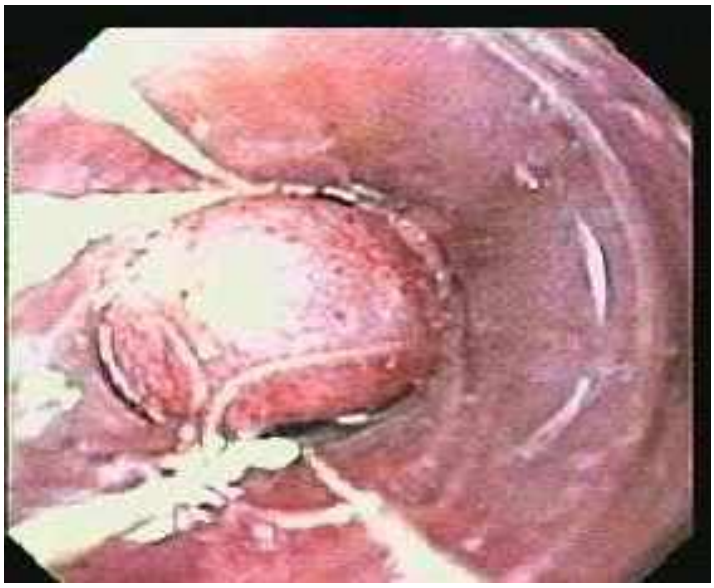

Fig 12: Endoscopic Banding

After intubation the device is kept in forward only mode. Once varix is identified, the tip is pointed towards it and continuous suction is applied to the varices till it is filled in the cap. Smooth movement left and right will help it. Once the red out sign appears, band can be fixed. Usually the procedure is performed by starting from gastro esophageal junction and proceeding upwards in a spiral fashion to avoid circumferential placement at the same level which would increase the risk of stricture.

In case of active bleeding the visual field is restricted due to the cylinder attachment which makes the technique difficult to performed

thus requiring active flushing with water and suction repeatedly. The rubber band should be delivered at a point on the varices but if it is missed, banding of normal mucosa is not harmful compared to sclerosant injection, which may cause serious side effects. If the bleeding point is not identified, then multiple banding devices can be used to place multiple bands at gastro esophageal junction checking that no subcardial prolongation occurs.

This might reduce torrential bleeding and the band can be fixed upward. After application of rubber bands over the esophageal varices, the ligated tissues with the rubber band will fall off within 10 days. The variceal sloughing causes shallow esophageal ulcers at the ligated sites while the esophageal varices reduce in size. Thought the ligation induced ulcers have a greater surface area, they are shallower and hence heal more quickly than that are caused by sclerotherapy.

Liquid diet is started for the first 12 hours and then the patient is advised to have soft foods. A recent study tells that patients who received pantoprazole after elective EVL found to have smaller post-banding ulcer than the other patients who received placebo therapy on follow up endoscopy. But the symptoms and ulcer number remained the same.

Eradication of the esophageal varices requires 2-3 sessions of endoscopic variceal ligation. De Franchis and Primignani conducted a

meta-analysis in 1999 included 14 articles in which the mean number of sessions to obliterate varices was reduced from5.4 in patients receiving sclerotherapy to 3.6 in patients receiving endoscopic variceal ligation. Both the time interval between the sessions should be noted to improve the efficacy of banding. Varices is said to be obliterated if they either disappear or unable to grasp.

Eradication can be obtained in about 89% of subjects although recurrence is common. The major disadvantages is higher incidence of recurrent varices. But these recurrent varices can be treated by ligation. Moreover recurrent varices do not increase the chance of rebleeding and do not cause endoscopic difficulties.

A study from Japan described that EVL performed once in two months is better that that is performed once in two weeks regarding variceal occurrence. Because rebleeding rate is significantly reduced who received endoscopic therapy at early and who achieves variceal obliteration in a shorter period. The incidence of bacteremia and infectious sequelae are less in EVL compared to sclerotherapy. Endoscopic band ligation is an alternate to sclerotherapy with fewer complications but there are the below list of complications:

Complications:

- Esophageal perforation
- Esophageal ulceration
- Retrosternal pain
- Transient dysphagia
- Gastropathy
- Esophageal strictures
- Ulcer bleeding
- Bacteremia

The advantage of EVL is the low rate of treatment induced complications. This is because the quantity of tissue ligated is limited by the design of the device resulting in fewer complications involving the esophageal wall.

Complications of EVL are either due to the ligation procedure or from the use of the overtube. Retrosternal pain, transient dysphagia occurs frequently in the immediate post ligation period and is considered as side effects rather than complications.

ESOPHAGEAL ULCERATION:

The band ligation usually produces small ulceration and rarely produces symptoms. They present as mucosal defects at the site of application of bands. Gimson *et al,* have reported esophageal ulceration in 35 out of 55 patients who had banding. 23 of them had small ulceration (size<5 mm) and 13 of them were having large (size >5 mm). Laine *et al*, has reported esophageal ulcerations producing rebleeding in 6% of patients who had undergone ligation. Steigmann has reported bleeding from ulcers in 12% of patients following ligation.

Young *et al*, compared the ulceration produced by ligation and sclerotherapy by means of scored ERCP cannula to measure the length, width and depth of ulcers in a randomized trial. Esophageal ligation produced shallow circular ulceration with large surface area that resolved in 14.4 days. Sclerotherapy produced linear, deep ulceration with a smaller surface area that resolved in 20.9 days.

Van Vlierberghe *et al*, reported early rebleeding after ligation in patients with Child-Pugh class C cirrhosis. This was attributed to the impaired clotting function as a result of liver disease and the greater size of the ulcers due to ligation.

ESOPHAGEAL STRICTURES:

Esophageal ulcerations leading into strictures are less common following ligation than with sclerotherapy. Laine et al, in a Meta analysis of 7 randomized trials involving 547 patients found esophageal strictures in 7 patients. Another study by Laine et al, demonstrated a significant reduction in stricture formation in ligation when compared to sclerotherapy (35%). Low rates of strictures formation have been reported by Baronicin due to ligation (11%) when compared to sclerotherapy. Steigmann and Sarin also reported a lower incidence of stricture formation following ligations (2% and 0 %).

ALTERATION OF ESOPHAGEAL MOTILITY:

In a study by Berner *et al*, 70% of patients had reported a transient dysphagia which lasted up to 24-72 hours after the procedure. This is due to the engorged banded varices. In a study conducted by Ming-Chih Hou *et al*, he compared the alteration of esophageal motility following sclerotherapy and ligation. He found that ligation produced a little change at 1 month or 3 month after eradication, while sclerotherapy produced a significant prolongation of transit time for 1 month after eradication which was reversible and improved after 3 months.

SYSTEMIC COMPLICATIONS:

Risk of bacteremia following ligation is 3-6% compared to sclerotherapy which is 5-53%. It is associated with fewer episodes of infectious sequelae such as spontaneous bacterial peritonitis or pneumonia.

Gin-Ho et al, reported that infectious sequelae due to sclerotherapy is about 20% compared to 1.9% in ligation. This is because the mechanical strangulation of varices during ligation obliterates the sub mucosal channels which diminish the entrance of bacteria into the blood stream. In a meta-analysis of 8 trials conducted by Laine et al, 7 out of 525 patients had pulmonary infection and 6 had bacterial peritonitis which was significantly lower comparing sclerotherapy.

OVERTUBE ASSOCIATED COMPLICATIONS:

In the past during endoscopic ligation using a single shot ligator the necessary repeated esophageal intubation with the scope is facilitated by a flexible plastic over tube passed over the endoscope. Majority of complications reported following ligation are associated with the use of over tube. Over tube injury to pharynx and proximal esophagus transient vocal cord paralysis, cricopharyngeal perforation, proximal esophageal perforation, varix rupture and free esophageal perforation have also been

reported. Mucosal injury had been reported in 72% of treatment sessions in one study. Massive bleeding has been reported distal to the over tube, probably due to blockage in venous outflow by the tube.

Esophageal perforation occurred with over the endoscope placement technique because of the large gap between the endoscope and the over tube which entrapped esophageal mucosa during the process of sliding the over tube over the endoscope during its insertion. Since the development of multiband ligator, not a single esophageal perforation has been reported. Banding is also associated with foods impaction resulting from a combination of lumen obstruction by banded varices and distal esophageal spasm.

OTHER ENDOSCOPIC OPTIONS:

TISSUE ADHESIVES (VARICEAL OBTURATION)

N-butyl-2-cyanoacrylate and isobutyl-2-cyanoacrylate have been used in the control of esophageal and gastric varices with control of bleeding in 80% of cases. Tissue adhesives were first used by Lunderquist in 1978 in treatment of varices. Cyanoacrylate is a hydrophilic tissue adhesive with a consistency similar to water. This is when added to blood rapidly polymerizes forming a solid cast of the injected vessels which results in rapid hemostasis of active bleeding and

prevents recurrence of bleeding. It has to be diluted with lipid based contrast agent to the dilution of about 2:1 ratio to delay the instantaneous polymerization within the injection syringe and needle.

Complications are due to embolisation of the glue producing cerebral stroke, pulmonary embolism, portal vein thrombosis, retro gastric abscess, visceral fistula and splenic infarction. Chances of damage to endoscope are high due to clogging of the accessory channel. There is also danger of eye injury due to accidental spraying of the cyanoacrylate.

ENDOLOOPS:

Endoloops are detachable nylon snares initially developed to control post polypectomy bleeding. This technique has been applied for control of bleeding from esophageal varices

MATERIALS AND METHODS

The study was conducted in the Department of General Surgery in collaboration with the Department of Medical Gastroenterology and Department of Vascular Surgery, Coimbatore medical College Hospital from June 2013- August 2015. This study was approved by the ethical committee of Coimbatore Medical College Hospital.

STUDY POPULATION:

50 patients with portal hypertension who were admitted during the study period of June 2013 - august 2015 in medicine and surgery wards, with the complaints of hematemesis and/or malena, who had grade 3 and 4 varices without gastric varices and other causes of upper GI bleeding in upper GI endoscopy were included in this study.

RANDOMISATION:

Every alternative patients presenting with above history is divided into 2 groups. One group is treated with esophageal banding and other group is treated with 3 % Sodium tetradecyl sulphate after getting informed and written consent from the patient.

INCLUSION CRITERIA:

- Age 21-70 years
- Both sexes
- Grade III and IV esophageal varices
- Patients complaining with hematemesis and/or malena
- Due to Portal Hypertension

EXCLUSION CRITERIA:

- Age <21 and >70
- Grade I and II varices
- Non portal hypertension causes of upper GI bleeding
- Prior history of endoscopic treatment and shunt operation for varices
- Presence of Hepatic Encephalopathy, Hepatorenal syndrome and life expectancy less than 48 hours
- Patients with positive serology for Hepatitis B (HbsAg) and C virus (anti HCV)

PROCEDURE:

Informed consent is obtained from the patient about the procedure. Patient is kept in NPO for 6 hours. Xylocaine spray is applied over the

posterior pharyngeal wall. Diagnostic endoscopy is performed. Presence of the culprit grade III and IV varices are identified and confirmed.

GROUP I:

They are subjected to esophageal variceal banding. Diagnostic endoscopy is performed and varices are identified. The distance is measured from the mouth by the markings in the endoscope. The endoscope is withdrawn and is loaded with ligation devices. Device is firmly attached to the scope and placed in neutral mode. Endoscopy with the loading devices is passed.

After intubation the device is kept in forward only mode. Once varix is identified, the tip is pointed towards it and continuous suction is applied to the varices till it is filled in the cap. Once the red out sign appears, band can be fixed starting from gastro esophageal junction and proceeding upwards in a spiral fashion.

GROUP II:

They are subjected to endoscopic sclerotherapy treatment. Diagnostic endoscopy is done and varices are identified. All visible varices are injected with 1-2 ml of 3% sodium tetradecyl sulphate below the bleeding site directly into the varices and the colour change in noted to confirm. Then the adjacent sub mucosa of the varices is injected with 1

ml of the sclerosant carefully upto 10 cm from Gastro esophageal junction proximally in a spiral fashion.

Care is taken that not more than 20 ml is injected in a single session to a patient. Three sessions were planned for every patient in an interval of 3 weeks. There after patients were reviewed once in a month for a period of three months. For each session the number of bands and the amount of sclerosant used are recorded. After the procedure all the patients are treated with beta blockers'.

During each visit, patients were assessed for complications such as retrosternal pain, esophageal ulcers, strictures, pleural effusion and mediastinitis.

Esophageal ulcers are defined as depression in the mucosal surface with overlying injury exudates. They were classified as superficial if shallow and less than 2 cm in diameter and as deep if more than 2 cm with shaggy border and a grayish necrotic base. Chest x ray was taken if the patients had persistent pain for detection of pleural effusion or mediastinitis if symptoms warranted.

Dysphagia was defined as difficulty in swallowing food and was graded into 4 grades:

- Grade I- Able to swallow both solid and liquid foods but was difficulty
- Grade II-Able to swallow liquid foods but not solid foods
- Grade III-Not able to swallow both solid and liquid foods
- Grade IV- Absolute dysphgia including inability to swallow saliva

Esophageal strictures were diagnosed if the patients reported with dysphagia and had evidence of narrowing by endoscopy and barium swallow. The efficacy was assessed in terms of

- Eradication of varices
- Number of sessions for variceal eradication
- Variceal recurrence
- Rebleeding episodes prior to eradication
- Associated complications

Variceal eradication was defined as the absence of visible variceal channels in the distal 5 cm of esophagus or presence of only mucosal tags. Variceal recurrence was defined as reemergence of variceal columns following previous complete eradication.

Control of active hemorrhage was defined as absence of clinically detectable upper GI bleeding for 48 hours after endoscopic variceal ligation was performed for active bleeding.

Failure of therapy was defined as recurrent variceal bleeding after three endoscopic treatment sessions or during the course of therapy.

STATISTICAL ANALYSIS:

Graph Pad Instat software was used for statistical analysis. Fisher's exact test was used for comparing the outcomes of endoscopic band ligation therapy and sclerotherapy. A p value of <0.05 was considered statistically significant.

RESULTS

In this study population, 29 males and 21 females have undergone treatment for varices and the majority were in the age group of 31-50 years

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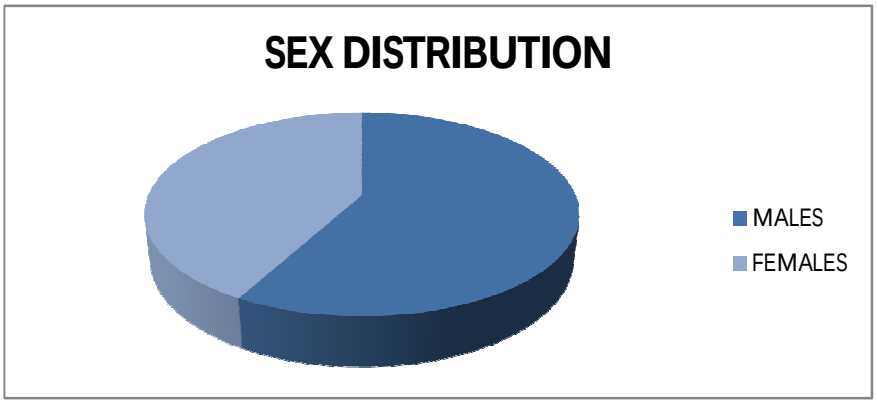

Fig13: Sex distribution of the study population

AGE AND SEX DISTRIBUTION

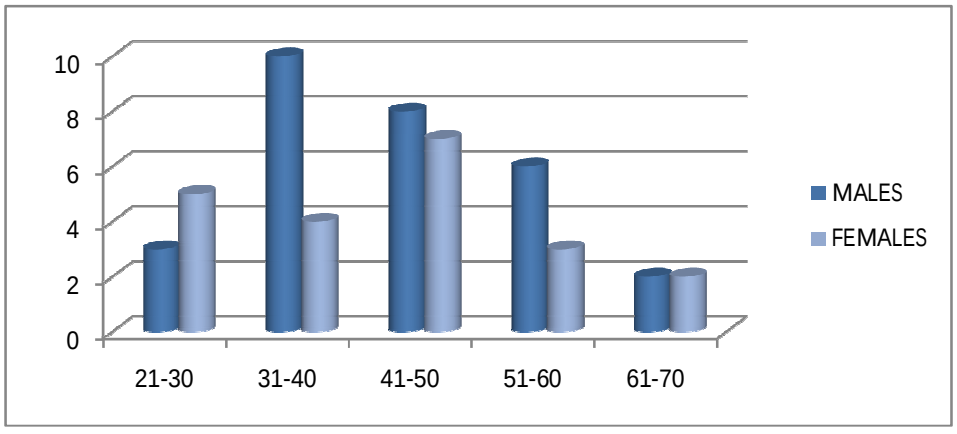

Fig14: Age and Sex Distribution of Study Populattion

AGE AND GRADE DISTRIBUTION

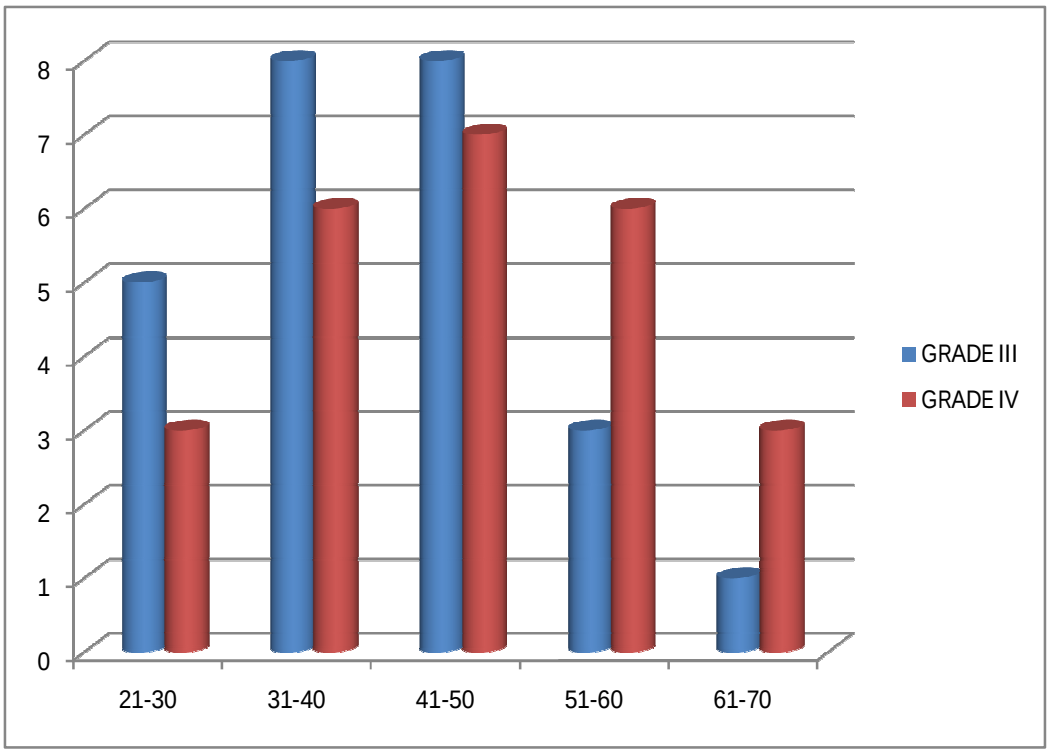

Fig15: Age and Variceal Grade Distribution of the Study Population

As the age progresses grade IV varices was more common than grade III

Study group randomization:

Table 1: Banding and sclerotherapy in bleeding varices

ACTIVELY BLEEDING VARICES	BANDING		SCLEROTHERAPY		TOTAL
	GRADE III	GRADE IV	GRADE III	GRADE IV	
+	6	4	5	7	22
-	7	8	7	6	28
TOTAL	**13**	**12**	**12**	**13**	**50**

22 cases presented with actve bleeding during the procedure and 28 patients were asymptomatic. Active bleeding was more common among male patients.

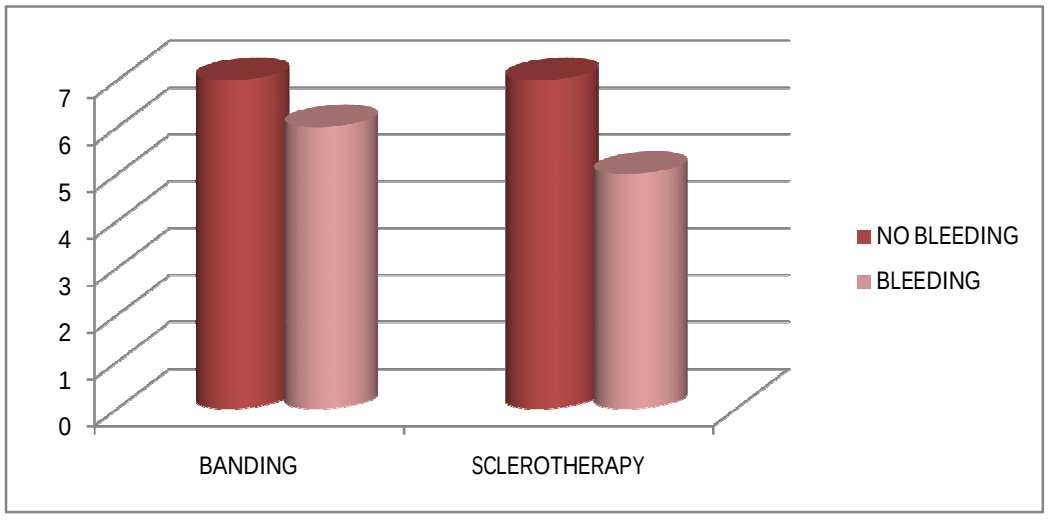

Fig 16: Randomization of Actively Bleeding Grade III Varices for Banding and Sclerotherapy

ACTIVE BLEEDING –GRADE IV VARICES

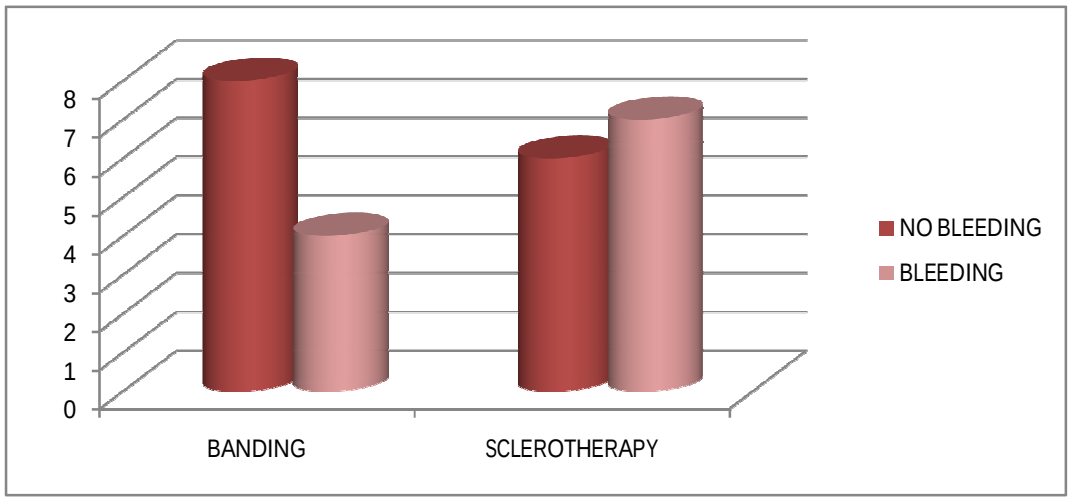

Fig 17:Randomization of active bleeding grade IV varices for banding and sclerotherapy

ACTIVE BLEEDING

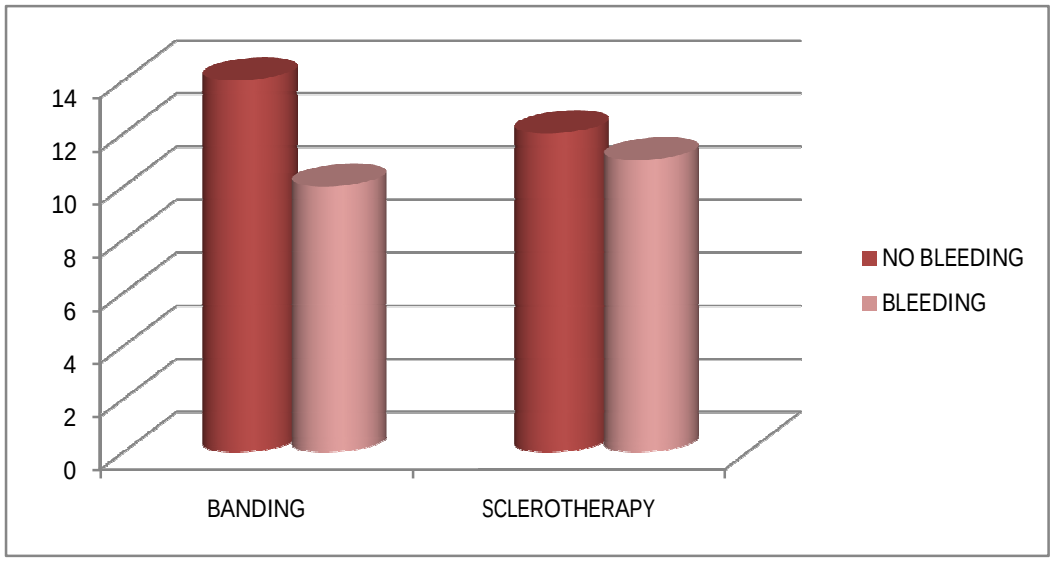

Fig18: Randomization of Actively Bleeding Varices for Banding and Sclerotherapy

NO OF BANDS & AMOUNT OF SCLEROTHERAPY USED

Table2: Number of Bands and Amount of Sclerotherapy used

GRADE	ACTIVE BLEED	TOTAL NO OF BANDS USED	TOTAL AMT. OF SCLEROSANT USED(ml)
III	+	52	168
	-	42	120
IV	+	44	196
	-	46	120
TOTAL		**194**	**532**

For grade III varices 94 bands and 188 ml of sclerosant used and for grade IV varices 100 bands and 216 ml of sclerosant used.

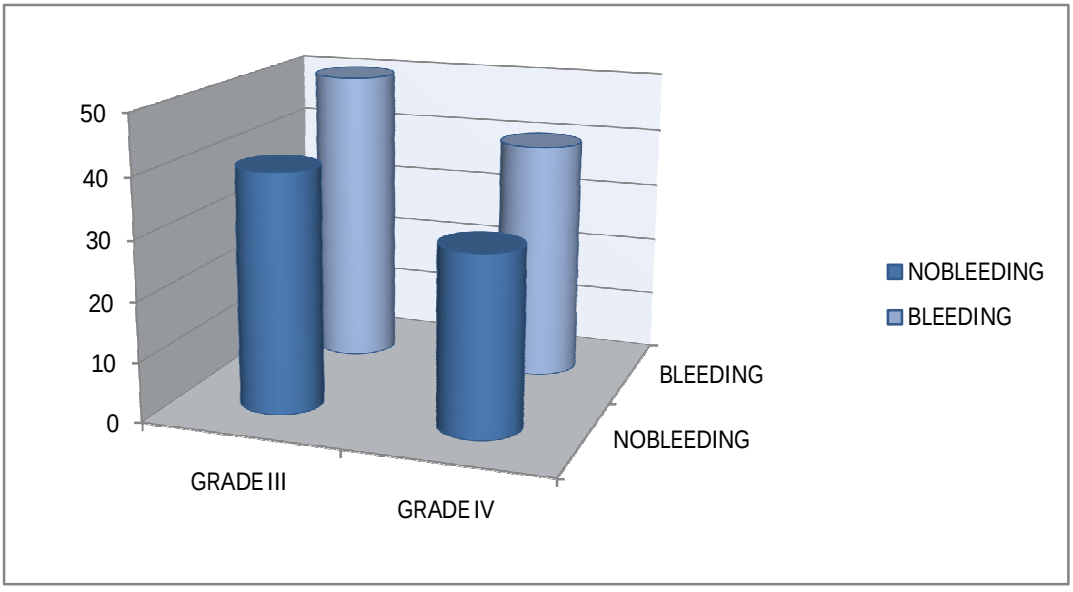

Fig19: Number of Bands used

AMOUNT OF SCLEROSANT USED

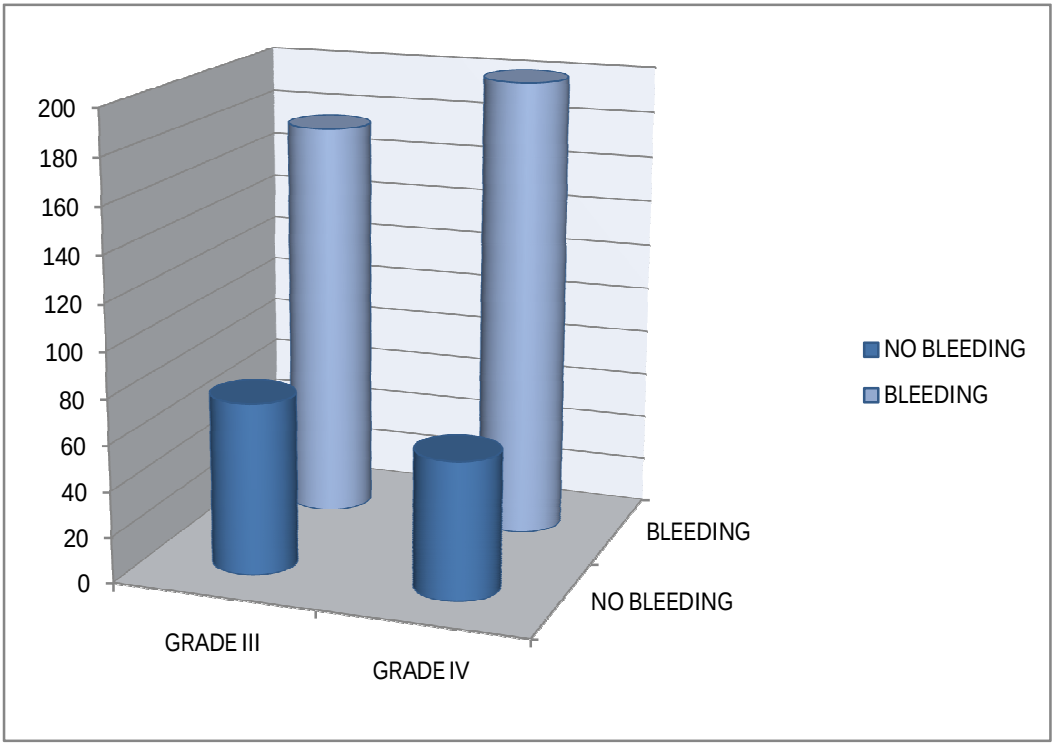

Fig20: Amount of Sclerosant used

Number of bands and amount of sclerotherapy used are more in active bleeding when compared to non bleeding varices.

COMPLICATIONS:

Table3: complications of banding ad sclerotherapy

COMPLICATIONS	BANDING				SCLEROTHERAPY			
	MALE		FEMALE		MALE		FEMALE	
		%		%		%		%
RETROSTERNAL PAIN	3	10	3	15	5	16.5	6	30
ODYNOPHAGIA	1	3.3	3	1	4	13.2	8	40
FEVER	1	3.3	1	5	5	16.5	6	30
TACHYCARDIA	0	0	2	10	3	10	5	25
ESOPHAGEAL ULCER	3	10	3	15	9	29.7	4	20
STRICTURE	0	0	0	0	1	3.3	0	0
BLEEDING	2	6.6	1	5	1	3.3	0	0
FAILURE	0	0	0	0	2	6.6	3	15

Males tolerated the procedure when compared to females.

RETROSTERNAL PAIN:

TABLE 4: comparison of retrosternal pain in banding and sclerotherapy

RETROSTERNAL PAIN	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	6	11	17
ABSENT	19	14	33
TOTAL	**25**	**25**	**50**

Though retrosternal pain is seen in patients who underwent sclerotherapy association was not statistically significant.(p value-0.2321)

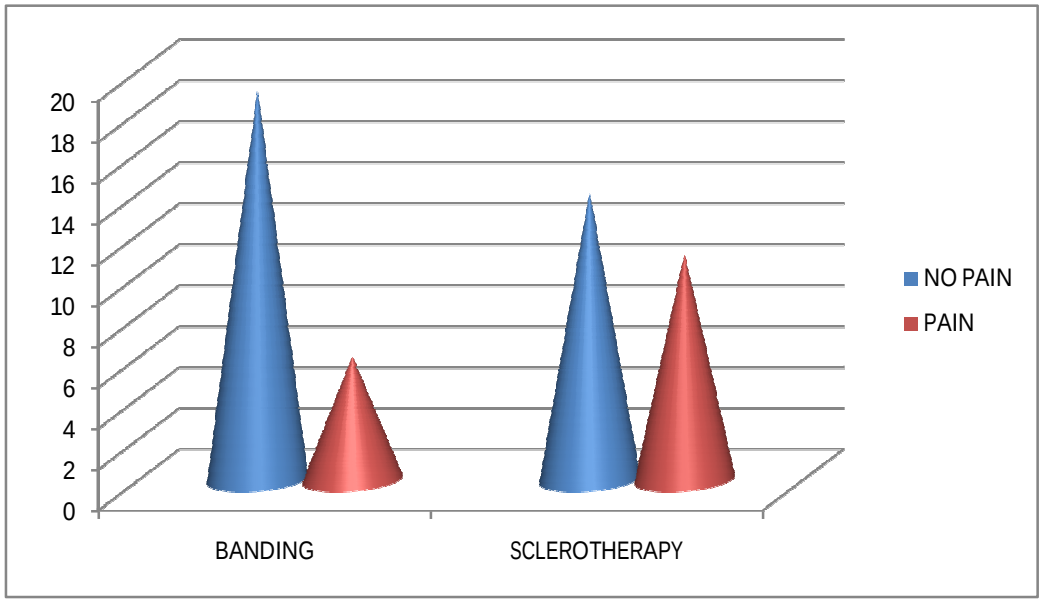

Fig 21: Comparison of Retrosternal Pain in Banding and Sclerotherapy

ODYNOPHAGIA:

TABLE5:Comparison of odynophagia in banding and sclerotherapy

ODYNOPHAGIA	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	4	12	16
ABSENT	21	13	34
TOTAL	**25**	**25**	**50**

Twelve patients in sclerotherapy group had odynophagia. However there is no statistically significant difference between two groups. (p value-0.0322)

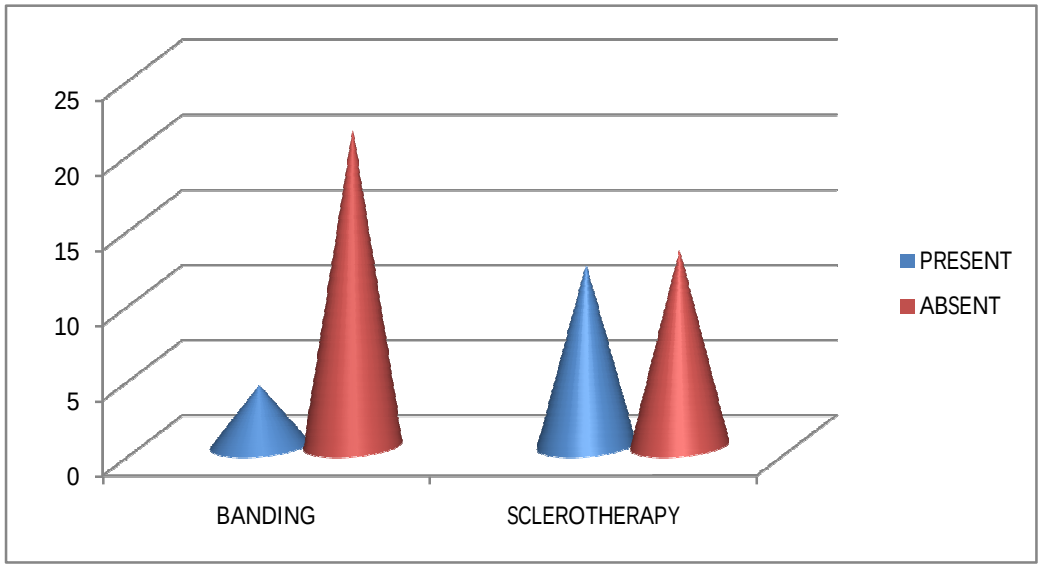

Fig22: Comparison of Odynophagia in Banding and Sclerotherapy

FEVER:

Table 6: Comparison of fever in banding and sclerotherapy

FEVER	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	2	11	13
ABSENT	23	14	47
TOTAL	**25**	**25**	**50**

Fever is most common in patients who had sclerotherapy while only two patients developed fever following banding and this is statistically significant (p value 0.0083)

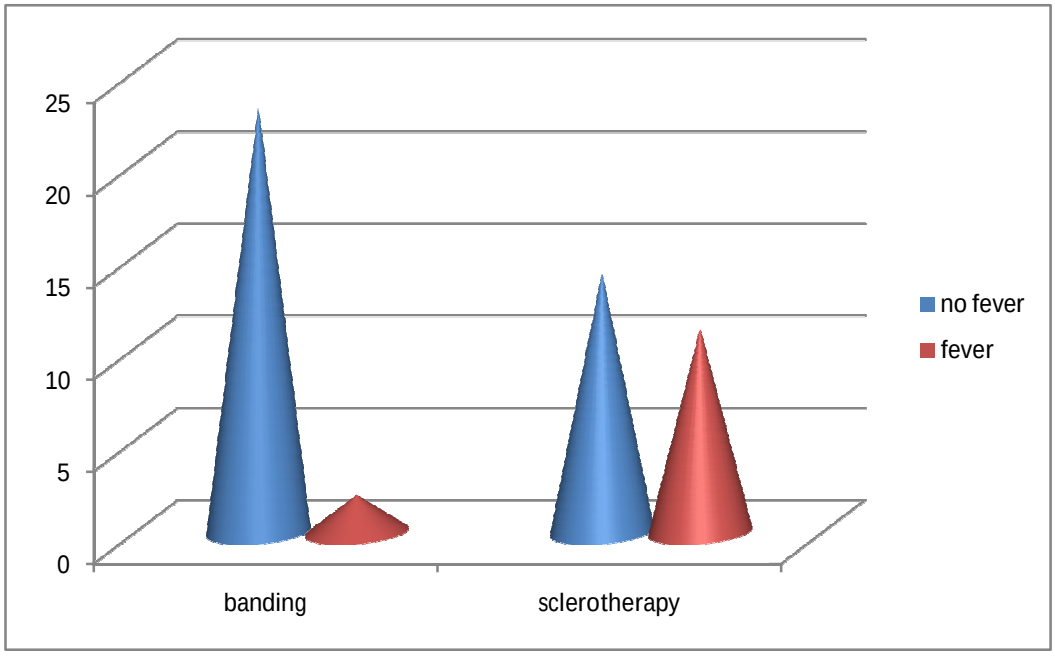

Fig23: Comparison of Fever in Banding and Sclerotherapy

TACHYCARDIA

Table 7: comparison of tachycardia in banding and sclerotherapy

TACHYCARDIA	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	2	8	10
ABSENT	23	17	40
TOTAL	**25**	**25**	**50**

The two study groups did not have statistically significant difference in tachycardia

(p value 0.0738)

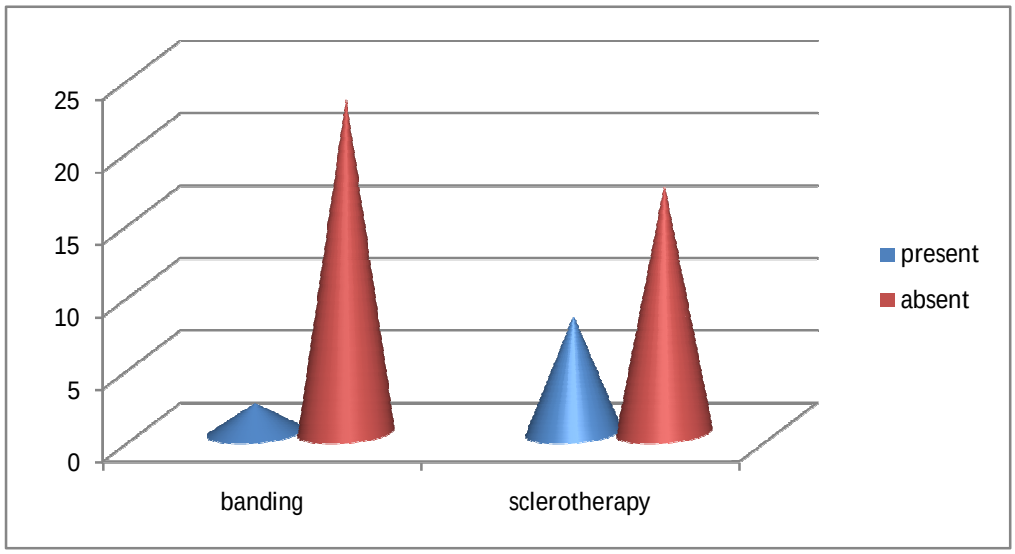

Fig24: Comparison of Tachycardia in Banding and Sclerotherapy

ESOPHAGEAL ULCER:

Table 8: comparison of ulcer in banding and sclerotherapy

ESOPHAGEAL ULCER	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	6	13	19
ABSENT	19	12	31
TOTAL	**25**	**25**	**50**

Esophageal ulcer was seen more in patients who underwent sclerotherapy but the association was not statistically significant (p value 0.0792)

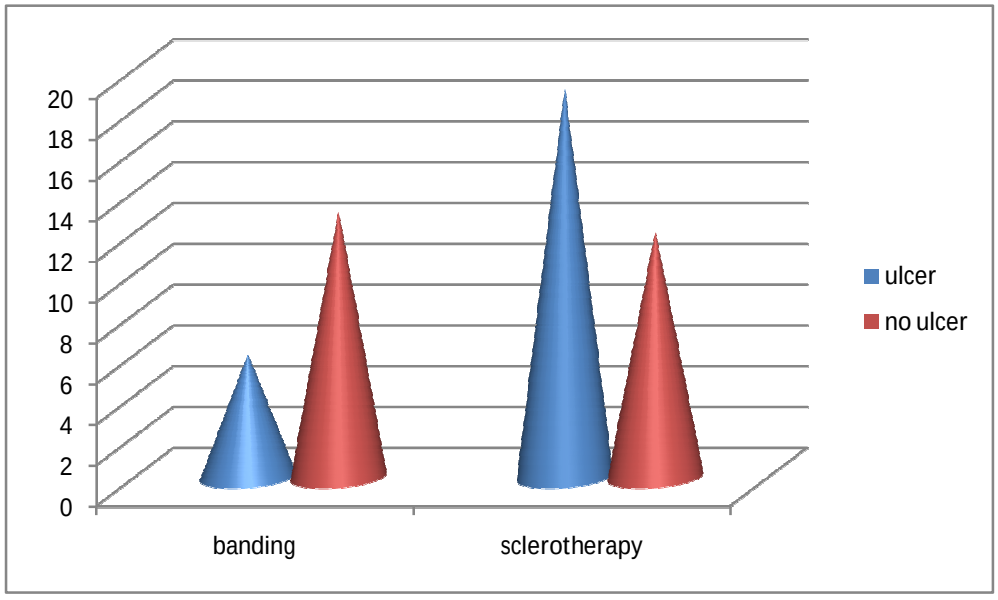

Fig25: Comparison of Esophageal ulcer in Banding and Sclerotherapy

ESOPHAGEAL STRICTURE:

Table 9: comparison of stricture in banding and sclerotherapy

STRICTURE	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	0	1	1
ABSENT	25	24	49
TOTAL	**25**	**25**	**50**

Only those patients who underwent sclerotherapy developed esophageal stricture, still it is not statistically significant (p value 0.3124)

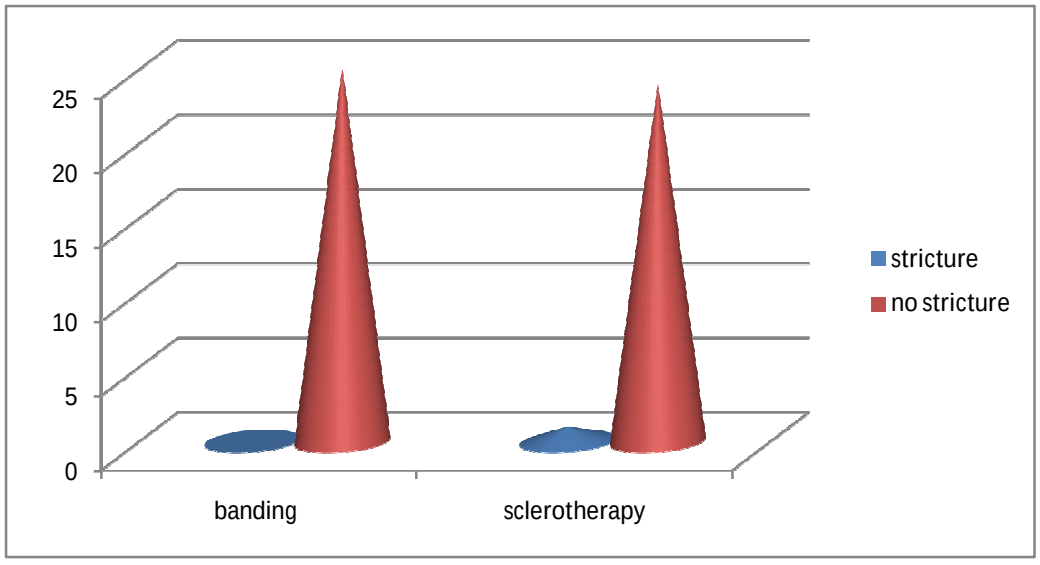

Fig26: Comparison of Stricture in Banding and Sclerotherapy

REBLEEDING

REBLEEDING	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	3	1	4
ABSENT	22	24	46
TOTAL	**25**	**25**	**50**

Rebleeding was more common in patients who underwent banding.However this difference was not statistically significant(p value 0.6022)

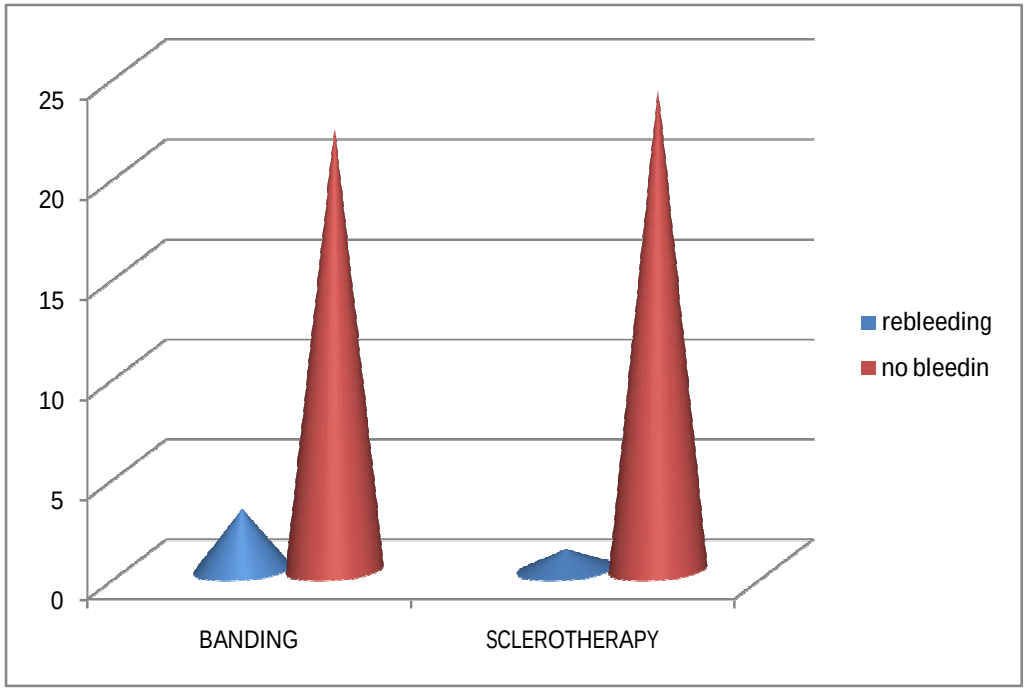

Fig27: Comparison of Rebleeding in Banding and Sclerotherapy

FAILURE

Table 11: comparison of failure in banding and sclerotherapy

FAILUR	BANDING	SCLEROTHERAPY	TOTAL
+	0	6	6
_	25	19	44
TOTAL	**25**	**25**	**50**

Failure is more common in patients who underwent sclerotherapy (p value 0.0223)

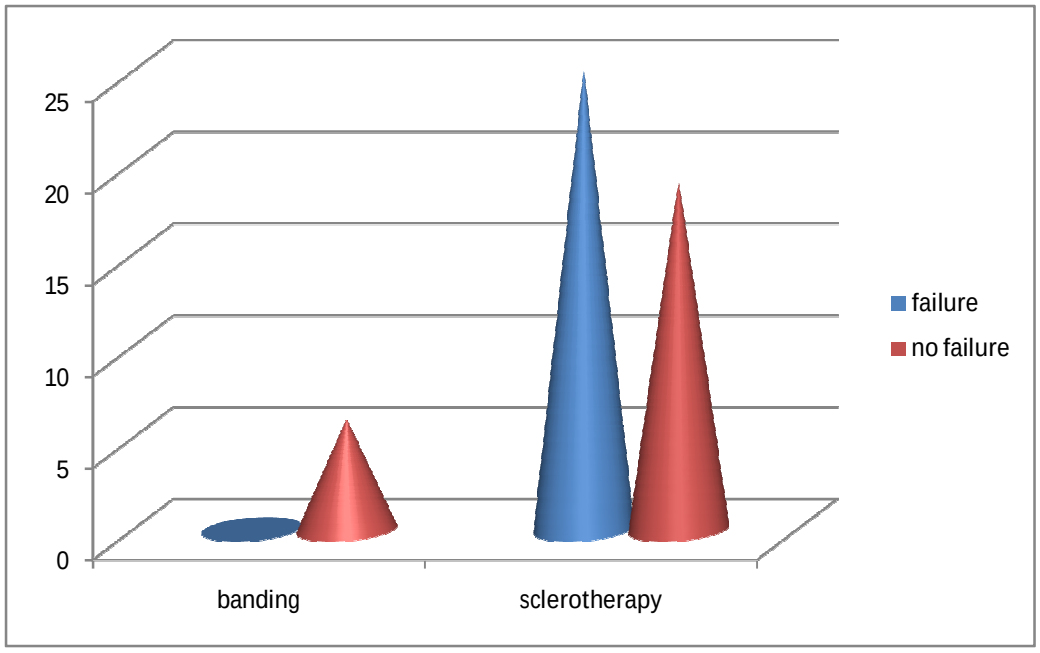

Fig28: Comparison of Failure in Banding and Sclerotherapy

DISCUSSION

The improvement in the results of the treatment of the variceal bleeding might be attributed to better clinical management of the above patients. Although in most of the studies performed, sclerotherapy is found to be inferior to band ligation for primary and secondary prophylaxis for variceal bleeding and also with lot of complications compared to banding some studies suggest that both are equally efficacious in the treatment of the esophageal varices. In 1986, Steigmann and his colleagues introduced band ligation which acts by mechanical action by causing strangulation of the variceal cord resulting in necrosis and scar formation 7-10days later. The difference in the technique is provided by number of bands used. Up to 10 bands can be used in a single session.

Since this procedure is easy to perform, results are often reproducible without variations. The difficult is that ligation of small varices is tedious. But in case of variceal injection of sclerotherapy, which was the first endoscopic treatment used approximately 50 years before the band ligation, there are numerous variations including the type of sclerossant, concentration of the agent, sclerosing technique, injected volume and location of the sclerosant (paravariceal and intravariceal combined) which is the reason for heterogeneous results of sclerotherapy

presented in different publication. And also this technique requires more experience and significant skill of the endoscopist and hence this technique is more operator dependent technique rather than banding. Hence the concept of combining ligation with sclerotherapy by employing ligation when the varices are large and converting to sclerotherapy when the varices becomes smaller has been put forward to maximize the benefits of both the technique and minimize the complications associated with each other.

But most of the studies which compared ligation and sclerotherapy with ligation alone showed no greater benefits.[26-28] A total of 40 patients included in the present study, 50% of the patients are in the age group of 31-50 years. Cirrhosis was the most common cause for portal hypertension. This was followed by non cirrhotic portal fibrosis and extra hepatic portal vein obstruction.

In the present study, only the patients who had either grade III or grade IV varices at presentation are included. Since patients with grade I and grade II were not considered for ligation because of technical difficulty in banding.

Most of the patient had cirrhosis had their etiology while the others had extra hepatic portal venous obstruction.

CONTROL BLEEDING:

A Metanalysis is done comparing the use of band ligation and 3% sodium tetradecyl sulphate and was published in 2006 and consists a total of 11 studies with a total of 1320 patients. The efficacy of endoscopic band ligation for initial hematemesis was found to be an average of 98% while that of the endoscopic sclerotherapy was found to be an average of 96%. Despite the better results obtained in the control of bleeding in band ligation than sclerotherapy, there is no difference in the mortality noted. In the present study, the efficacy of banding is 96% and that of sclerotherapy is 89%. This excellent control of varicel bleeding is comparable to other reports has been mentioned by many authors that during active bleeding, presence of fresh blood and blood clots obscures the vision leading to difficulty in banding. In this study about 25 patients had active bleeding. In that 10 were subjected to banding, 10 were subjected to sclerotherapy. About 2 patients had rebleeding in banding and 3 patients had rebleeding who were subjected to sclerotherapy.

VARICEAL ERADICATION:

Several studies on ligation have reported successful eradication of varices in 50-95% of patients. Steigmann et al, reported an eradication rate of 52% with ligation with a median of 5 treatment sessions and 15 ligation per patient.

Laine et al , in their study of 39 patients who underwent ligation reported 58% of eradication with a median of 4 treatment sessions at an average of 3.9 bands at each session.[21] Gimson et al, reported zn eradication rate of 70% in patientswho underwent ligation with a median of 3.4 endoscopic sessions. Sarles et al, reported 28% obliteration rate. In another study, variceal obliteration was achieved in 54% of patients who were treated with sodium tetradecyl sulphate. Despite these old studies, new studies such as Bhargava et al, reported eradication in 87% of patients at a median of 6 5 endoscopic sessions and also showed 88% eradication rate in use of sodium tetradecyl sulphate.[30]

The King,s college study reported a satisfactory eradication of esophageal varices by the use of banding with less complication than sclerotherapy although much of the the complications are strictures. A study conducted by Grimson and Ramage et al , with an aim to find whether endoscopic variceal ligation is more effective in eradicating varices than sclerotherapy showed that both the techniques were effective in controlling the bleeding(92% for banding and 91% for sclerotherapy).variceal obliteration was not achieved in some patients in each group(3% banding and 6% sclerotherapy). Though there was no significant difference between the above two techniques in eradication, ligation achieved more quickly than sclerotherapy. Thus in newer studies,

the efficacy of both band ligation and sclerotherapy in eradicating the varices have increased a lot. This have multiband ligating devices rather than single band ligating devices. In the present study of about 50patients, 25 were treated by endoscopic variceal leading and other 25 by endoscopic sclerotherapy (3% sodium tetradecyl sulphate), the eradication of varices by banding was 100% while that of sclerotherapy was only 80%.

As the most of the previous studies, the present study also suggests that the endoscopic variceal banding is superior to sclerotherapy in the eradication of varices.

COMPLICATIONS:

Most of the studies suggest that the main advantage of ligation over sclerotherapy is the low rate of complications. Laine et al, have reported complication in 24% of patients who had ligation. ALTraif et al, have reported a complication rate of 60% using sclerotherapy.[28] In the present study, the complications' were found in 30 to 50 patients among whom the majorities were esophageal ulceration, retrosternal pain, odynophagia, fever and tachycardia. Similar observations were made out in most of the studies. The patients who had active bleeding during the procedure had more complication rate.

ESOPHAGEAL ULCER:

Esophageal ulcer was the commonest complication following sclerotherapy in most of other studies. The occurrence of post sclerotherapy ulceration was attributed to the higher volume of sclerosant per session, shorter interval between sclerotherapy sessions, and higher volume of sclerosant and nature of the sclerosant used. In case of banding large superficial ulcerations are common due to necrosis. Esophageal ulceration was reported in 36% of patients who had undergone ligation by Gimson et al, in the study of 54 patients.[21] Korula et al, had also shown similar reports- 70% of patients who had sclerotherapy. Blenkinsopp et al , showed that diluting sodium tetradecyl sulphate from 3 % to 1 % reduce the rate of ulceration with only a minimal decrease in efficacy. Westaby et al m compared the effect of sclerotherapy at one weekly interval and at three weekly intervals and found that ulceration is common at one weekly interval.

In present study, the esophageal ulceration is found to be the most common complication of both banding and sclerotherapy. 6 out of 25 patients had esophageal ulceration who underwent variceal banding while 13 patients of 25 had esophageal ulceration who underwent sclerotherapy. Thus 25% of patients who had banding and about 50% of patients who had sclerotherapy developed esophageal ulceration.

All ulceration was found to be superficial without bleeding. The higher incidence of ulceration in the sodium tetradecylsulphate group was probably due to concentration used (3%) and also due to ulcerogenic property of sodium tetradecyl sulphate.

RETROSTERNAL PAIN:

Transient retrosternal pain following sclerotherapy can be due to mediastinitis and due to esophagitis. Korula et al, reported an incidence of 24% with 1.5% sodium tetradecyl sulphate.[31] In a study done by Lebski et al, the banding is associated with about 40% of patients developed retrosternal pain among which 6 out of 25 patients who underwent banding and 11 of 25 patients who underwent sclerotherapy had retrosternal pain.

ODYNOPHAGIA:

In a study Berner et al, 75% of patients had reported a transient dysphagia which lasted upto 24-72 hours after the procedure. This is due to the engorged banded varices. Bargava et al, noted that dysphagia significantly common with sclerotherapy with sodium tetradecylsulphate. In the present study also, odynophagia is more commonly seen in sodium tetradecyl sulphate. In the present study also, odynophagia is more commonly seen in sodium tetradecyl sulphate group than banding group.

The difference was statiscally significant. Edema and inflammation around the ulcer contributes to the narrowing of esophagus. This explains why dysphagia is more common in use of sclerotherapy because of its ulcerogenic property. Hence around 4 patient of 25 who underwent banding and 12 of 25 patients who underwent sclerotherapy had dysphagia with the significant ‘p’

FEVER:

In most of studies, fever lasting for 24-48 hours after sclerotherapy and banding occurred in 20-40% of patients. Fever usually subsided spontaneously. In the present study, fever was seen in 8 % of patients who underwent banding and 44% of patients who underwent sclerotherapy with significant ‘p’ value. Most of the other studies have shown similar report with present study.

TACHYCARDIA:

Tachycardia following banding and sclerotherapy could be due to febrile spikes or anxiety by the procedure. Kumar at al, reported tachycardia in 48% of patients with 3% sodium tetradecyl sulphate. In the present study 32% of patients who underwent sclerotherapy had tachycardia while only 8 % patients who underwent banding had tachycardia.

ESOPHAGEAL STRICTURE:

Esophageal strictures are due to healing of deep esophageal ulceration. Bargava et al , reported an incidence of 27% of strictures with 1.5% sodium tetradecyl sulphate. Sorensen used 3% sodium tetradecyl sulphate and reported strictures in 35% of patients. He attributed this higher rate to frequent sclerotherapy sessions. Most of other studies reported strictures rate ranging from 1-20%. Laine et al, in a meta-analaysis of 7 randomized trials involving 547 patients found esophageal strictures in 7 patients. Another studyby Laine et al, demonstrated significant reduction in stricture formation in ligation. Low rates of stricture formation have been reported by Baronicin et al, due to ligation compare to sclerotherapy. Steigmann and Sarin also reported a lower incidence os stricture following ligation. In the present study only one patient developed stricture following sclerotherapy after 4 months . while no patient develop stricture following banding. This is due to proper banding technique in spiral fashion and restricting sclerotherapy to proximal OG junction.

SUMMARY

This study is conducted prospectively on total of 50 patients with bleeding esophageal varices from September 2013 to august 2015 with the prime aim of evaluating the efficacy and safety of endoscopic variceal banding and endoscopic sclerotherapy.

- In this study cirrhosis is the most common etiology of portal hypertension accounting for 90% of study population.
- 60 % of the population were in 31-50 years of age group
- In this study more than 70% of old age people presented grade IV than grade III varices
- In this study 45% of patients had signs of active bleeding
- In actively bleeding varices, sclerotherapy has a little added advantage over banding because of the technical difficulty of the banding due to obscured field.
- In this study about 194 bands and 532 ml of sclerosant were used wth a mean of 7.76 bands and 21.2 ml of sclerosant per person.

- In active bleeding varices the mean number of bands and the amount of sclerotherapy is more compared to that of mean of number of bands and amount of sclerotherapy in non-bleeding patients.

- In this study more number and percentage of females had complications compared to males though the study population of females is less

- 24% of patients had retrosternal pain in banding while 44%of patients in sclerotherapy had retrosternal pain

- 16% of patients with banding and 48% of patients with sclerotherapy had odynophagia

- 22% of patients with banding and 44% of patients with sclerotherapy had fever

- 32% of patients with sclerotherapy had tachycardia. No patients with banding complained of tachycardia

- 24%of patients with banding and 52% of patients with sclerotherapy had esophageal ulceration

- 4% of patients with sclerotherapy developed stricture. No patients with banding developed stricture over the follow up period.

- 12% of patients with banding and 3% of patients with sclerotherapy had rebleeding during and after the procedures
- 20% of patients with sclerotherapy had recurrences of varices while no recurrence was seen in banding for the follow up period
- ‘P’ value is significant in complications such as odynophagia,fever and recurrence

CONCLUSION

- Both banding and 3% sodium tetradecyl sulphate are equally effective in controlling acute variceal hemorrhage among which sclerotherapy had a small advantage and also in preventing rebleeding.
- Both banding and sclerotherapy are effective in eradicating varices but banding is more efficacious
- Both banding and sclerotherapy have their side effects but sclerotherapy has more frequent and dreaded complications.
- Hence banding is superior to sclerotherapy both in efficacy and safety

ABBREVIATIONS

DSRS	-	Distal Splenorenal shunt
EBL	-	Endoscopic band ligation
EVL	-	Endoscopic variceal ligation
GIT	-	Gastrointestinal tract
GEJ	-	Gastroesophageal junction
HVPG	-	Hepatic venous pressure gradient
PTFE	-	Polytetrafluroethylene
STD	-	sodium tetradecyl sulphate
TIPS	-	Transjugular intrahepatic portosystemic shunt

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ANNEXURE-1

PROFORMA

Serial Number:

Name: Sex:

Age: Ward:

Hospital no:

Address:

D.O.A D.O.D D.O.S

I. PRESENTING COMPLAINTS

Hemetemesis:

Malena:

Jaundice:

Fever:

Abdominal pain:

Abdominal distension:

Altered sensorium:

Pedal edema:

Drug intake:

II. PAST HISTORY

Hemetemesis: Hypertension:

Malena: PREVIOUS TREATMENT

Jaundice:

Alcoholism:

III. EXAMINATION

Built and Nourishment : Icterus:

Mental status:

Cyanosis:

Pallor:

Clubbing:

Lymphadenopathy:

E/o Liver failure:

PR: Temp: RR: BP:

P/A:

Distension: Abdominal veins:

Liver: Spleen:

Ascitis:

Respiratory system:

CVS:

IV:INVESTIGATIONS:

Hb:	Platelet count:
TC:	Peripheral smear:
DC:	
Blood Urea:	Blood sugar:
Na/k:	Hbs Ag:
Sr.Bilirubin:	SGPT:
SGOT:	Sr.Alk.PO4:

USG of Abdomen:

V.TREATMENT

Date:

Grade of columns:

No. of. Columns:

No. of bands applied:

Amount of sclerosant injected:

VI: COMPLICATIONS:

Retrosternal Pain:

Odynophagia:

Fever:

Tachycardia:

Esophageal ulcer:

Esophageal perforation:

Pleural Effusion:

Mediastinitis:

Rebleed:

FOLLOWUP

SESSION	1	2	3	FOLLOW UP	
DATE					
GRADE					
NO.OF.COLUMNS					
NO.OF.BANDS APPLIED					
AMOUNT OF SCLEROSANT USED					
COMPLICATIONS					

MASTER CHART

S.NO	NAME	AGE	SEX	IP.NO	DIAGNOSIS	GRADE	BLEEDING	NO OF BANDS PER SESSION			RETROSTERNAL PAIN	ODYNOPHAGIA	FEVER	TACHYCARDIA	ULCER	STRICTURE	REBLEEDING	FAILURE
								I	II	III								
1	Amaravathy	36	F	52652	Cirrhosis	III	+	6	3	1	+	+	-	-	+	-	-	-
2	Srinivasan	50	M	56321	Cirrhosis	III	-	5	1	-	-	-	-	-	-	-	-	-
3	Neelaveni	55	F	59165	EHPVO	IV	-	5	1	-	-	-	-	-	-	-	-	-
4	Ganesh	48	M	48562	Cirrhosis	III	+	6	2	1	-	-	-	-	-	-	-	-
5	Rukmani	48	F	52689	Cirrhosis	IV	+	6	3	2	+	-	+	+	+	-	-	-
6	Ramathal	48	F	54265	Cirrhosis	IV	+	6	4	1	-	-	-	-	-	-	-	-
7	Kasi	51	M	58659	Cirrhosis	IV	-	5	1	-	-	-	-	-	-	-	-	-
8	Bannari	42	F	54896	Cirrhosis	III	-	5	1	-	-	-	-	-	-	-	-	-
9	Karpagam	28	F	42658	Cirrhosis	III	+	6	2	-	-	-	-	-	-	-	-	-
10	Kalyani	24	F	54896	EHPVO	III	-	5	1	-	-	-	-	-	-	-	-	-
11	Kanmani	69	F	56325	Cirrhosis	IV	-	5	1	-	-	-	-	-	-	-	-	-
12	Palanisami	52	M	52566	Cirrhosis	IV	+	5	1	-	-	-	-	-	-	-	-	-
13	Rajendran	42	M	54236	Cirrhosis	III	+	6	2	-	-	-	-	--	-	-	-	-
14	Arul	40	M	51123	Cirrhosis	IV	-	6	4	1	+	-	-	-	+	-	+	-
15	Kumar	40	M	52654	Cirrhosis	III	-	5	1	-	-	-	-	-	-	-	-	-
16	Sivagami	50	F	52314	Cirrhosis	IV	+	6	3	2	+	-	-	+	+	-	+	-
17	Kalimuthu	35	M	54865	Cirrhosis	III	-	5	1	-	-	-	-	-	-	-	-	-
18	Raman	55	M	52231	Cirrhosis	IV	-	5	-	-	+	-	+	-	+	-	-	-
19	Neelamani	40	F	51265	Cirrhosis	III	+	5	4	-	+	-	-	-	+	-	-	-
20	Ravikumar	53	M	53265	Cirrhosis	IV	-	5	-	-	-	-	-	-	-	-	-	-
21	Jaganathan	36	M	51426	Cirrhosis	III	-	5	1	-	-	-	-	-	-	--	-	-
22	Vimal	22	M	59624	EHPVO	IV	-	5	1	-	-	-	-	-	-	-	-	-
23	Vinoth	38	M	57465	Cirrhosis	IV	-	5	1	-	-	-	-	-	-	-	-	-
24	Karthikeyan	26	M	51232	EHPVO	IV	+	6	2	1	-	-	-	-	-	-	+	-
25	Lakshmi	55	F	52539	Cirrhosis	III	-	5	1	-	-	-	+	-	-	-	-	-

S.NO	NAME	AGE	SEX	IP.NO	DIAGNOSIS	GRADE	BLEEDING	SCLEROSANT USED PER SESSION			RETROSTERNAL PAIN	ODYNOPHAGIA	FEVER	TACHYCARDIA	ULCER	STRICTURE	REBLEEDING	FAILURE
								I	II	III								
1	Rajan	65	M	51236	Cirrhosis	III	+	16	9	8	+	-	+	-	+	-	-	-
2	Rangammal	60	F	51254	Cirrhosis	IV	-	13	8	5	+	+	+	+	-	-	-	-
3	Chandran	41	M	52526	Cirrhosis	IV	-	10	8	6	-	-	-	-	+	-	-	-
4	Zubair	40	M	53265	Cirrhosis	III	+	15	11	8	+	+	-	-	+	-	-	-
5	Ganesh	39	M	58965	Cirrhosis	IV	+	10	10	8	-	-	-	-	-	-	-	-
6	Palani	58	M	54126	Cirrhosis	IV	-	11	5	2	-	-	-	-	-	-	-	-
7	Marathal	43	M	52369	Cirrhosis	IV	+	15	8	5	+	+	+	+	+	-	-	+
8	Muniyandi	50	M	54756	Cirrhosis	III	_	11	5	2	+	+	+	+	-	-	-	-
9	Lakshmi	60	F	51512	Cirrhosis	IV	-	16	7	4	+	+	+	+	-	-	-	-
10	Poongothai	33	F	52645	Cirrhosis	III	-	10	3	0	-	+	+	+	-	-	-	-
11	Natraj	43	M	54754	Cirrhosis	IV	+	17	9	6	+	+	-	-	+	-	-	-
12	Sundaram	50	M	52516	Cirrhosis	IV	+	11	7	6	-	-	-	-	+	-	-	-
13	Sugapriya	24	F	53146	EHPVO	IV	_	16	10	9	-	+	-	-	+	-	-	-
14	Venugopal	65	M	58951	Cirrhosis	III	+	13	10	5	-	-	+	+	+	+	-	-
15	Sarojini	40	F	52468	Cirrhosis	IV	_	10	2	0	+	+	-	-	-	-	-	-
16	Selval	65	F	52516	Cirrhosis	III	-	8	2	3	-	-	-	-	-	-	-	=
17	Poovathal	50	F	54326	Cirrhosis	IV	+	10	6	4	+	+	+	+	-	-	-	+
18	Sekar	33	M	59564	Cirrhosis	IV	-	14	9	3	-	-	-	-	+	-	-	-
19	Eswari	40	F	54216	Cirrhosis	III	+	9	5	1	-	-	+	-	-	-	-	-
20	Jeyalakshmi	46	F	52365	Cirrhosis	III	-	12	5	3	+	+	-	-	-	-	-	-
21	Ravi	48	M	56896	Cirrhosis	III	-	15	10	9	+	+	-	-	+	-	+	+
22	Natraj	52	M	55565	Cirrhosis	IV	-	14	12	5	-	-	+	+	+	-	-	+
23	Niwas	26	M	52231	EHPVO	IV	-	11	3	1	-	-	+	-	-	-	-	-
24	Vasanth	36	M	54465	Cirrhosis	III	+	10	11	7	-	-	-	-	+	-	-	-
25	Thenmozhi	28	F	53326	EHPVO	III	-	10	3	1	-	-	-	-	+	-	-	-

xggpjy;gotk;

bgah; :

ghypdk; :

Kfthp : taJ :

muR nfhit kUj;Jtf; fy;Yhpapy; bghJ mWit rpfpr;ir Jiwapy; gl;l nkwgogg[gapYk; khzth; k.etpd] mth;fs; nkwbfhs;Sk; "**A COMPARATIVE STUDY OF ESOPHAGEAL VARICES – BANDING VS SCLEROTHERAPY**" vdw nrhjidapd; brayKiw kwWk; midj;J tpgu';fisak; nflLfbfhz;lJld; vdJ midj;J renjf';fisak; bjspthpggLj;jpfbfhz;nld;vdgij bjhptpj;Jf;bfhs;fpnwd;

ehd;,e;j Ma;tpy;KG rkkjj;JlDk; Ra rpe;jidak;lDk; fye;J bfhs;s rkkjpf;fpnwd;

,e;j Ma;tpy; vd;Dila midj;J tpgu';fSk; ghJfhf;fggLtJld; ,jd; Kotpfs; Ma;tpjHpy; btspaplggLtjpy;vdf;F ve;j Mlnrgidak;k;,y;iy vdgij bjhptpj;Jf;bfhs;fpnwd; ve;j neuj;jpYk;,e;j Ma;tpy;,Ue;J tpyfpfbfhs;s vdf;F chpik cz;L vdgijak;mwpntd;

,lk; :

njjp : **ifbahggk;/ nuif**